WEST

Help | Logout | Interrupt

Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences | Cases

Search Results -

Term	Documents
((10 OR 9) AND 4).USPT.	0
((L10 OR L9) AND L4).USPT.	0

Database: US Patents Full-Text Database
US Pre-Grant Publication Full-Text Database
JPO Abstracts Database
EPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L11

Refine Search

Recall Text | Clear

Search History

DATE: Saturday, October 19, 2002 Printable Copy Create Case

********** STN Columbus **********

FILE 'MEDLINE'
FILE 'JAPIO'
FILE 'BIOSIS'
FILE 'SCISEARCH'
FILE 'WPIDS'
FILE 'CAPLUS'
FILE 'EMBASE'
=> s il-13 or IL13 or interleukin 13 or interleukin-13
L1 108760 IL-13 OR IL13 OR INTERLEUKIN 13 OR INTERLEUKIN-13

=> il and antibod?
L2 2282 IL AND ANTIBOD?

=> s il-13 n10 antibod?
L3 0 IL-13 N10 ANTIBODI?

=> s il 13 n10 antibod?
L4 0 IL 13 N10 ANTIBODI?

=> s il 13 n10 antibod?
L5 0 IL 13 N10 ANTIBOD?

=> s il 13 10a antibod?
L6 0 IL 13 10A ANTIBOD?

=> s il 13 10a antibod?
L7 0 IL-13 10A ANTIBOD?

L8 0 INTERLEUKIN 13 10A ANTIBOD?

=> interleukin 13 10a antibod?
L9 0 INTERLEUKIN 13 10A ANTIBOD?

=> interleukin-13 10a antibod?
L10 0 INTERLEUKIN-13 10A ANTIBOD?

=> d his

(FILE 'HOME' ENTERED AT 20:18:07 ON 19 OCT 2002)

FILE 'MEDLINE, JAPIO, BIOSIS, SCISEARCH, WPIDS, CAPLUS, EMBASE' ENTERED AT 20:18:20 ON 19 OCT 2002
L1 108760 S IL-13 OR IL13 OR INTERLEUKIN 13 OR INTERLEUKIN-13
L2 2282 L1 AND ANTIBOD?
L3 0 S IL-13 N10 ANTIBODI?
L4 0 S IL 13 N10 ANTIBODI?
L5 0 S IL 13 N10 ANTIBOD?
L6 0 S IL 13 10A ANTIBOD?
L7 0 S IL-13 10A ANTIBOD?
L8 0 INTERLEUKIN 13 10A ANTIBOD?
L9 0 INTERLEUKIN-13 10A ANTIBOD?
L10 0 INTERLEUKIN? WITH ANTIBOD?
L11 0 INTERLEUKIN? WITH ANTIBOD?
L12 2098 INTERLEUKIN? WITH ANTIBOD?
L13 0 INTERLEUKIN'13 WITH ANTIBOD?
L14 0 S INTERLEUKIN'13 ANTIBOD?
L15 0 S IL'13 ANTIBOD?
L16 0 S IL'13 ANTIBODIES
L17 0 S IL'13 ANTIBODY
L18 46 IL-13 ANTIBODY
L19 21 ANTIBODIES TO IL-13
L20 41 ANTIBODY TO IL-13
L21 41 S ANTIBODY TO IL-13
L22 12 S ANTIBODY TO INTERLEUKIN-13
L23 11 S ANTIBODIES TO INTERLEUKIN-13

=> dup rem l18
PROCESSING COMPLETED FOR L18
L24 11 DUP REM L18 (35 DUPLICATES REMOVED)

=> dup rem l19
PROCESSING COMPLETED FOR L19
L25 7 DUP REM L19 (14 DUPLICATES REMOVED)

=> dup rem l20
PROCESSING COMPLETED FOR L20
L26 16 DUP REM L20 (25 DUPLICATES REMOVED)

=> dup rem l22
PROCESSING COMPLETED FOR L22
L27 12 DUP REM L22 (0 DUPLICATES REMOVED)

=> dup rem l21
PROCESSING COMPLETED FOR L21
L28 16 DUP REM L21 (25 DUPLICATES REMOVED)

=> dup rem l23
PROCESSING COMPLETED FOR L23
L29 11 DUP REM L23 (0 DUPLICATES REMOVED)

=> d l29 ibib abs 1-11

L29 ANSWER 1 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:636225 CAPLUS
DOCUMENT NUMBER: 135:206417
TITLE: Interleukin 13 gene single nucleotide polymorphisms and their use in diagnosis of immune disorders
INVENTOR(S): Panteliadis, Panagiotis
PATENT ASSIGNEE(S): Royal Brompton and Harefield NHS Trust, UK
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001062925	A2	20010830	WO 2001-GB707	20010220
WO 2001062925	A3	20011220		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-118018P P 19990201
AB Materials and methods are disclosed for modulating proliferation of cell type associated with Hodgkin's disease through inhibition or of IL-13 and components in IL-13 associated signal transduction pathways. Methods to identify inhibitors, compns. comprising the inhibitors, and methods using the inhibitors to treat Hodgkin's disease are also disclosed

L29 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:475556 CAPLUS
DOCUMENT NUMBER: 133:86260
TITLE: Method for diagnosing, imaging, and treating tumors bearing interleukin 13-specific receptors
INVENTOR(S): Debinski, Waldemar; Connor, James R.
PATENT ASSIGNEE(S): The Penn State Research Foundation, USA
SOURCE: PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000040254	A1	20000713	WO 2000-US149	20000105

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-115149P P 19990107
AB Methods are provided for diagnosing, imaging, and treating tumors bearing interleukin 13-specific receptors. Methods are also provided for identifying compds. that bind to interleukin 13-specific receptors. Compns. and kits are provided for diagnosing, imaging, and treating tumors bearing interleukin 13-specific receptors.

EP 1140167 A1 20011010 EP 2000-901390 20000105
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO
JP 2002534395 T2 20021015 JP 2000-592019 20000105
PRIORITY APPLN. INFO.: US 1999-226794 A 19990107
WO 2000-US149 W 20000105
AB Disclosed is a method of inhibiting the growth of tumors bearing IL-13-specific receptors. Included among this class of tumors is glioblastoma multiforme (GBM), a rapidly progressing brain tumor for which there is currently no effective treatment available. In the disclosed method, a chimeric cytotoxin comprising an IL-13 receptor binding moiety and a cytotoxic moiety is delivered into a mammalian subject having a tumor bearing IL-13 specific receptors. All studied human GBM specimens abundantly express the IL-13-specific tumor.
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:180729 CAPLUS
DOCUMENT NUMBER: 128:256388
TITLE: Therapeutic molecules
INVENTOR(S): Nicola, Nicos Antony; Hilton, Douglas James; Zhang, Jian-Guo; Simpson, Richard John
PATENT ASSIGNEE(S): Amrad Operations Pty. Ltd., Australia; Nicola, Nicos Antony; Hilton, Douglas James; Zhang, Jian-Guo; Simpson, Richard John
SOURCE: PCT Int. Appl., 70 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9810038	A1	19980319	WO 1997-AU591	19970910
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GE, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9741049	A1	19980402	AU 1997-41049	19970910
PRIORITY APPLN. INFO.:			AU 1996-2262	19960910
			AU 1997-5374	19970227
			WO 1997-AU591	19970910

AB The present invention provides therapeutic mols. capable of interacting with interleukin-13 (IL-13) and to genetic sequences encoding these therapeutic mols. The IL-13-binding proteins (IL-13BP) bind to IL-13 with a greater affinity than sol. interleukin 13 receptor alpha1 chain (IL-13R alpha1), and have mol. wt. about 40 apprx. 60 kDa. The IL-13BP mols. of the present invention are useful in modulating the action of IL-13 in vivo, and for treating allergic reaction. Also disclosed are monoclonal antibody to IL-13BP, and transgenic animals comprising IL-13BP mutant gene.

L29 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:288048 CAPLUS
DOCUMENT NUMBER: 129:26848
TITLE: Regulation of interleukin-13 receptor constituents on mature human B lymphocytes
AUTHOR(S): Ogata, Haruki; Ford, Dwayne; Kouttab, Nicola; King, Thomas C.; Vita, Natalio; Minty, Adrian; Stoeckert, Johanna; Morgan, Deborah; Grasole, Christopher; Morgan, John W.; Manzel, Abby L.
CORPORATE SOURCE: Roger Williams Med. Cent., Brown Univ., Providence, [illegible]
[illegible]
AB Human B [illegible] receptors composed of the IL-4R alpha chain complexed with either the IL-13R alpha 1 or gamma c occur simultaneously within defined B cell populations. MENAs for all receptor constituents are increased subsequent to Ig sterilization alone, while maximal expression of IL-13R alpha 1 is more dependent upon co-stimulation of Ig and CD40 receptors. MRNA levels for IL-13R alpha 1 vary over a wider range subsequent to surface stimulation than other receptor components. Although gamma c is not bound to IL-13 on B cells under the conditions evaluated, it may influence IL-13 binding by competing with IL-13R alpha 1 for assocn. sequestration with the IL-4R alpha chain. IL-13R alpha 2 does not participate in the IL-13 receptor that is up-regulated upon activation of quiescent tonsillar B lymphocytes, although mRNA for the protein may be found in the centroblastic fraction of tonsillar cells.

L29 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:594756 CAPLUS
DOCUMENT NUMBER: 127:258660
TITLE: Cloning and expression of cDNA for interleukin-13 binding chain of IL-13 receptor, identification of inhibitors of binding, and treatment of Ig-mediated disease
INVENTOR(S): Collins, Mary; Donaldson, Debra; Fitz, Lori; Neben, Tamlyn; Whitters, Matthew; Wood, Clive
PATENT ASSIGNEE(S): Genetics Institute, Inc., USA
SOURCE: PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9731946	A1	19970904	WO 1997-US3124	19970228
W: AU, CA, JP, MX				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5710023	A	19980120	US 1996-609572	19960301
AU 9719801	A1	19970916	AU 1997-19801	19970228
US 6214559	B1	20010410	US 1997-841751	19970430
US 6248714	B1	20010619	US 1997-846340	19970430
US 6268480	B1	20010731	US 1997-846344	19970430
PRIORITY APPLN. INFO.:			US 1996-609572 A	19960301
			WO 1997-US3124 W	19970228

AB Polynucleotides encoding the IL-13-binding subunit of the IL-13 receptor and fragments thereof are disclosed. IL-13 receptor proteins, methods for their prodn., inhibitors of binding of IL-13 and its receptor and methods for their identification are also disclosed. Use of the inhibitors for treatment of Ig-mediated diseases such as allergies, asthma, lupus, etc. are further disclosed. Mouse and human IL-13 receptor IL-13 binding chain cDNAs are cloned and sequenced. A recombinant sol. IL-13 binding chain fused to an Ig was prepd. and shown to inhibit IL-13-stimulated B9 cell proliferation.

L29 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:465174 CAPLUS
DOCUMENT NUMBER: 127:107998
TITLE: Interleukin-13 receptor subunits of human, cDNAs encoding them, and their diagnostic and therapeutic uses
INVENTOR(S): Caput, Daniel; Ferrara, Pascual; Laurent, Patrick; Vita, Natalio
PATENT ASSIGNEE(S): Sanofi, Fr.; Caput, Daniel; Ferrara, Pascual; Laurent, Patrick; Vita, Natalio
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXX, 92
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9720926	A1	19970612	WO 1996-FR1756	19961107
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG				
CA 2238080	AA	19970501	CA 1996-2238080	19961023
AU 9672668	A1	19970515	AU 1996-72668	19961023
AU 718899	B2	20000420		
EP 940756	A1	19990414	EP 1996-934193	19961023
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 11514873	T2	19991221	JP 1996-516141	19961023
US 20020060332	A1	20020711	US 2001-36568	20011107
PRIORITY APPLN. INFO.:			AU 1995-72276	A 19951222
			AU 1996-2208	A 19960909
			WO 1996-AU668	W 19961023
			US 1998-51845	A1 19980629

AB The present invention relates generally to a novel hematopoietin receptor, NR4, which is the interleukin-13 receptor alpha-chain, or components or parts thereof and to genetic sequences encoding the same. The receptor mols. and their components and or parts and the genetic sequences encoding same of the present invention are useful in the development of a wide range of agonists, antagonists, therapeutics and diagnostic reagents based on ligand interaction with its receptor. Mouse and human NR4 cDNA sequences are included.

L29 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:425227 CAPLUS
DOCUMENT NUMBER: 127:30144
TITLE: Interleukin-13 receptor alpha-chain protein NR4, mouse and human cDNA sequences, and applications in assays for asthma and allergy therapeutics and diagnostics
INVENTOR(S): Willson, Tracy; Nicola, Nicos A.; Hilton, Douglas J.; Metcalf, Donald; Zhang, Jian Guo
PATENT ASSIGNEE(S): Amrad Operations Pty. Ltd., Australia; Willson, Tracy; Nicola, Nicos A.; Hilton, Douglas J.; Metcalf, Donald; Zhang, Jian Guo
SOURCE: PCT Int. Appl., 92 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9715663	A1	19970501	WO 1996-AU668	19961023
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG				
CA 2238080	AA	19970501	CA 1996-2238080	19961023
AU 9672668	A1	19970515	AU 1996-72668	19961023
AU 718899	B2	20000420		
EP 940756	A1	19990414	EP 1996-934193	19961023
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 11514873	T2	19991221	JP 1996-516141	19961023
US 20020060332	A1	20020711	US 2001-36568	20011107
PRIORITY APPLN. INFO.:			AU 1995-72276	A 19951222
			AU 1996-2208	A 19960909
			WO 1996-AU668	W 19961023
			US 1998-51845	A1 19980629

AB The present invention relates generally to a novel hematopoietin receptor, NR4, which is the interleukin-13 receptor alpha-chain, or components or parts thereof and to genetic sequences encoding the same. The receptor mols. and their components and or parts and the genetic sequences encoding same of the present invention are useful in the development of a wide range of agonists, antagonists, therapeutics and diagnostic reagents based on ligand interaction with its receptor. Mouse and human NR4 cDNA sequences are included.

L29 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible]
INVENTOR(S): [illegible]
PATENT ASSIGNEE(S): [illegible] American ENA
SOURCE: PCT Int. Appl., [illegible] pp.
CODEN: PIXXD2
[illegible]

PATENT INFORMATION

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9629417	A1	19960926	WO 1996-US3486	19960315

W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE,
ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT,
LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE,
SG, SI
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR,
IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN
US 5614191 A 19970325 US 1995-404685 19950315
CA 2215122 AA 19960926 CA 1996-2215122 19960315
AU 9653110 A1 19961008 AU 1996-53110 19960315
AU 714541 B2 20000106
EP 1007696 A1 20000614 EP 1996-909693 19960315
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, FI
JP 2000511042 T2 20000829 JP 1996-528499 19960315
US 5919456 A 19990706 US 1997-821840 19970321
PRIORITY APPLN. INFO.: US 1995-404685 A 19950315
WO 1996-US3486 W 19960315

AB A method and compns. are provided for specifically delivering an effector mol. to a tumor cell. The method involves providing a chimeric mol. that comprises an effector mol. attached to a targeting mol. that specifically binds an interleukin-13 (IL-13) receptor and contacting a tumor cell with the chimeric mol. The target moiety of the the chimeric mol. may consist of IL-13, an anti-IL-13 receptor antibody, or circularly permuted IL-13, the effector moiety may be a cytotoxin (Pseudomonas exotoxin, Diphtheria toxin, ricin, or abrin), label, radionuclide, drug, liposome, ligand, or antibody. Thus, recombinant DNA technol. was used to produce single-chain fusion proteins human IL-13 (or its circularly permuted analog) to either of 2 mutant forms of Pseudomonas aeruginosa exotoxin A. Circularly permuted IL-13 is a deriv. in which the normal N- and C-termini are linked via the Gly-Gly-Ser-Gly linker peptide, and the bond between Gly-43 and Met-44 is broken, thereby yielding cpIL-13 in which Met-44 is the new N-terminus and Gly-43 is the new C-terminus. PE38QQR is a truncated form of Pseudomonas exotoxin composed of amino acids 253-364 and 381-608, the lysine residues at positions 509 and 606 are replaced by Gln and at 613 is replaced by Arg, P34L is a full-length Pseudomonas exotoxin with a mutated and inactive binding domain where amino acids 57, 246, 247, and 249 are replaced by glutamate. The fusion protein IL-13-PE38QQR targets the IL-13 receptor on human renal cells and is high cytotoxic to cells expressing high nos. of IL-13 receptor. Because resting or activated immune cells or bone marrow cells are not sensitive to IL-13-toxin, this toxin is useful for the treatment of renal carcinoma cells without being cytotoxic to normal immune cells. Human glioma cells, medulloblastoma, and Kaposi's sarcoma are also highly sensitive to the IL-13-PE38QQR as well as to the immunotoxins cpIL-13-PE38QQR, IL-13-PE4E, and cpIL-13-PE4E.

L29 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:352885 CAPLUS
DOCUMENT NUMBER: 125:31750
TITLE: IL-13 released by and localized in human basophils
AUTHOR(S): Li, Huamin; Sim, Tommy C.; Alam, Rafeul
CORPORATE SOURCE: Department Internal Medicine, University Texas Medical Branch, Galveston, TX, 77555, USA
SOURCE: Journal of Immunology (1996), 156(12), 4833-4838
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We and others have shown that human basophils can synthesize and release IL-3, and A23187 in a dose-dependent manner. PBMC, neutrophils, and eosinophils isolated from the same donors did not release IL-13 after anti-IgE stimulation. The anti-IgE-induced basophil IL-12 synthesis could be enhanced by IL-3 preincubation (with and without IL-3 preincubation anti-IgE-induced IL-13 prodn. was 227 and 42 pg/106 basophils, resp.). PBMC produced a significant amt. of IL-13 upon stimulation with PHA, but a low level of IL-13 in response to A23187 and or PMA. Eosinophils and neutrophils did not produce IL-13 when cultured with A23187, IL-3, and anti-Fc epsilon RI alpha. This is the first demonstration of IL-13 prodn. by basophils. Our data suggest that basophils, in addn. to secreting mediators, can represent an important source of proallergic cytokines.

L29 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:30117 CAPLUS
DOCUMENT NUMBER: 126:103048
TITLE: Interleukin-13, in combination with anti-interleukin-12, increases graft prolongation after portal venous immunization with cultured allogeneic bone marrow-derived dendritic cells
AUTHOR(S): Ourezynski, Reginald M.; Cohen, Zane; Fu, Xin-Ming; Hua, Zeng; Sun, Yongliang; Chen, Zhiqi
CORPORATE SOURCE: Departments Surgery and Immunology, University Toronto, Toronto, M5G 2C4, Can.
SOURCE: Transplantation (1996), 62(11), 1592-1600
CODEN: TRPLAU; ISSN: 0041-1337
PUBLISHER: Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Portal venous (pv) transfusion before transplant with large nos. (100 times 106) of irradiated multiple minor histoincompatible spleen cells (B10.Br) augments allogeneic skin graft survival in C3H mice. We have shown in earlier studies that this is correlated with preferential activation for prodn. of type 2 cytokines (interleukin [IL]-4 and IL-10) and decreased prodn. of type 1 cytokines (IL-2 and interferon [IFN] gamma). We have also shown that recombinant rhIL-12, in assocn. with anti-IL-10 monoclonal antibody can reverse in vivo the graft prolongation afforded by pv immunization and the altered cytokine prodn. that follows. Adoptive transfer of inhibition of graft rejection is possible at early times after pv immunization, using plastic adherent cells obtained from the liver of treated mice. We show that within 4 days of pv immunization, dendritic cells (NLDC-145+) isolated from the thymus, mesenteric lymph node (MLN), and spleen of mice receiving MHC-incompatible cells grafts (C3H with C57BL/6), can transfer skin graft prolongation to naive C3H recipients. Moreover, 8 times 106 cultured dendritic cells derived from 10-day cultures of C57BL/6 bone marrow also confer increased graft survival after pv immunization, but not after i.v. immunization. Once again, increased graft survival with cultured dendritic cells was assocd. with polarization of T cells that were isolated from treated mice to produce IL-4 and IL-10 on restimulation in vitro. Graft survival and polarization in cytokine prodn. was further enhanced by simultaneous administration of anti-IL-12 monoclonal antibody, rIL-13, or more significantly, a combination of anti-IL-12 and rIL-13. These alterations were assocd. with persistence of donor cells in various tissues of recipient mice, as assessed using polymerase chain reaction for expression of Y-chromosome in female recipients of male bone marrow. Our data suggest that a combined strategy of donor-specific immunization before transplant and manipulation of cytokine levels in vivo may prove an effective regimen in the induction of unresponsiveness in transplant recipients.

=> d l28 ibib ab 1-16

L28 ANSWER 1 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:696137 CAPLUS
DOCUMENT NUMBER: 137:231354
TITLE: Method for constructing expression cassette of a chimeric interleukin 13 (IL-13) vaccine and therapeutic uses
INVENTOR(S): Aiman, Claire; Crowe, James Scott; Ellis, [illegible]

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

GE, GH,
GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU,
TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH,
CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: GB 2001-5360 A 20010303

AB The present invention provides a method for constructing expression cassette of a chimeric interleukin 13 (IL-13) vaccine in which the sequence of the predicted antigenic loops has been taken from murine IL-13, and the sequence of the predicted structural (predominantly helical) regions has been taken from human IL-13. The present invention relates to an isolated polypeptide useful for immunization against self-antigens. In particular the invention relates to a self-protein that is capable of raising auto-antibodies when administered in vivo. The invention particularly relates to rendering human cytokines immunogenic in humans. The invention further relates to pharmaceutical compns. comprising such compds. and their use in medicine and to methods for their prodn.

REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L28 ANSWER 2 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:343936 BIOSIS
DOCUMENT NUMBER: PREV200200343936
TITLE: A monoclonal antibody to mouse IL-13 inhibits acute asthma response.
AUTHOR(S): Yang, Gaoyun (1); Emmell, Eva (1); Sheafy, Dave (1); Griswold, Don (1); Li, Li (1)
CORPORATE SOURCE: (1) Centocor, Inc., 200 Great Valley Parkway, Malvern, PA, 19355 USA
SOURCE: FASEB Journal, (March 20, 2002) Vol. 16, No. 4, pp. A672.
http://www.fasebj.org/. print.
Meeting Info.: Annual Meeting of the Professional Research Scientists on Experimental Biology New Orleans, Louisiana, USA April 20-24, 2002
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English

AB Mouse interleukin 13 (IL-13) is a pleiotropic cytokine mainly produced by Th2 cells. Over-expression of IL-13 in the lung or treatment of mice with recombinant IL-13 intranasally induced airway hyperresponsiveness (AHR), mucus gland hyperplasia, eotaxin production, pulmonary eosinophila and subepithelial fibrosis. On the other hand, blocking IL-13 using either the IL-13 receptor-Ig fusion protein or polyclonal antiserum in asthmatic mice significantly inhibited AHR, mucus production, airway inflammation an fibrosis. These results suggested that IL-13 is a key player in asthma pathogenesis, therefore IL-13 specific monoclonal therapy could provide therapeutic potential on asthma. To prove the concept, we have developed a rat anti-mouse IL-13 neutralizing monoclonal antibody and tested its effects on OVA induced acute asthma responses in mice. IL-13 was up-regulated in the lung during OVA induced asthma responses. When administered at the challenge stage, the anti-IL-13 monoclonal antibody significantly inhibited AHR, goblet cell hyperplasia and mucus production. Furthermore, the antibody treatment also inhibited the production IL-5, IL-6, and eotaxin in the lung. These results clearly demonstrated that IL-13 plays an important role in asthma responses, and suggest that a monoclonal ***antibody*** to ***IL*** - ***13*** would be an effective therapeutic agent in the treatment of asthma.

[illegible] ANSWER 3 OF 16 [illegible] COPYRIGHT 2002 [illegible]

[illegible] PATENT NO. KIND DATE APPLICATION NO. DATE

[illegible] CORPORATE SOURCE: [illegible] Yorktown, VA, USA
SOURCE: JOURNAL OF RHEUMATOLOGY, (2002) Mar; 29 (3) 454-61.

FILE SEGMENT: Priority Journals
ENTRY MONTH: 200209
ENTRY DATE: Entered STN: 20020324
Last Updated on STN: 20020925
Entered Medline: 20020924

AB OBJECTIVE: To investigate the physiology of interleukin 13 (IL-13) in rheumatoid arthritis (RA) and the effects of tumor necrosis factor (TNF) antagonists (etanercept) on the distribution of IL-13 in patients with RA.

METHODS: We measured cytokine levels in RA sera (pre/post etanercept), RA synovial fluid (SF), osteoarthritis (OA) SF, and normal human sera by ELISA. Detection of IL-13 was not influenced by rheumatoid factor, as revealed in spike recovery and isotype antibody control studies. Biological activity of IL-13 in RA SF was studied using dendritic cell (DC) progenitors that develop into mature DC with IL-13 and with neutralizing ***antibodies*** to ***IL*** - ***13***. The modulation of IL-13 by etanercept was compared to that of IL-6 and monocyte colony stimulating factor (M-CSF). The effect of etanercept on the ability of RA sera to promote DC growth was studied using DC progenitors. RESULTS: IL-13 was increased in RA sera versus normal sera, OA SF, and RA SF. Relative to OA SF and normal sera, RA SF was enriched in IL-13. The IL-13 contained in RA samples was biologically active, prompting DC growth from progenitors. Circulating DC growth activity was strongly reduced by anti-TNF therapy. Whereas decreases in DC growth factors including IL-13 and IL-6 occurred with etanercept therapy and were associated with clinical improvement, concurrent increases in circulating M-CSF (a non-DC, monocyte-specific growth factor) were noted. CONCLUSION: The increase of biologically active IL-13 in RA supports the concept that IL-13 regulates immune cell (including dendritic cell) activity and indicates how the varied anatomical distribution of cytokines may play a role in the RA disease process. The differential regulation of circulating IL-13 and M-CSF levels by TNF antagonists further implies discrete roles in the TNF-cytokine network in RA.

L28 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:360036 CAPLUS
DOCUMENT NUMBER: 134:365710
TITLE: Modulating IL-13 activity using mutated IL-13 molecules that are antagonists or agonists of IL-13
INVENTOR(S): Puri, Raj K.; Oshima, Yasuo; Joshi, Bharat H.
PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA
SOURCE: PCT Int. Appl., 129 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001034645	A2	20010517	WO 2000-US31044	20001110
WO 2001034645	A3	20020307		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
AU 2001018993 A1 20010606 AU 2001-18993 20001110
PRIORITY APPLN. INFO.: US 1999-165236P P 19991110
WO 2000-US31044 W 20001110

residues at positions 112, 110, 109, 92, 69, or 66 are mutated to a neutrally charged residue, or one with a charge opposite to the charge of the residue found at that position in native IL-13, provided that the residue at position 112 of the mol. is not neg. charged. The agonists can be used as more potent agents to provoke an effect provided by IL-13 in particular, the agonists can be used as reagents in the maturation of monocytes into dendritic cells, or to pretreat bone marrow stem cell donors to reduce graft vs. host disease in the recipient of the stem cells. Finally, the invention provides IL-13 receptor binding mols. with affinity for the IL-13 receptor at least about 3 times greater than that exhibited by wild-type IL-13. Also provided are methods and compns. for specifically delivering an effector mol. to a tumor cell by chimeric mols. comprising the effector mol. and an IL-13 receptor binding mol., and pharmaceutical compns. comprising such chimeric mols.

L28 ANSWER 5 OF 16 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2001255129 MEDLINE
DOCUMENT NUMBER: 21217071 PubMed ID: 11316662
TITLE: Decreased steroid responsiveness at night in nocturnal asthma. Is the macrophage responsible?
AUTHOR: Kraft M; Hamid Q; Chrousos G P; Martin R J; Leung D Y
CORPORATE SOURCE: Departments of Medicine and Pediatrics, National Jewish Medical and Research Center, Division of Pulmonary Sciences and Critical Care Medicine, University of Colorado Health Sciences Center, Denver, Colorado, USA. kraftm@njc.org
CONTRACT NUMBER: AR-41256 (NIAMS)
HL03343 (NHLBI)
HL36577 (NHLBI)
RR-00051 (NCRR)
SOURCE: AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE, (2001 Apr) 163 (5) 1211-25.
Journal code: 9421642. ISSN: 1073-449X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010611
Last Updated on STN: 20010611
Entered Medline: 20010607

AB As peripheral blood mononuclear cells from patients with nocturnal asthma (NA) exhibit reduced steroid responsiveness at 4:00 A.M. as compared with 4:00 P.M., we hypothesized that NA is associated with increased nocturnal airway cell expression of GRbeta, an endogenous inhibitor of steroid action. Ten subjects with NA and seven subjects with nonnocturnal asthma (CNA) underwent bronchoscopy with bronchoalveolar lavage (BAL) at 4:00 P.M. and 4:00 A.M. BAL lymphocytes and macrophages were incubated with dexamethasone (DEX) at 10(-5) to 10(-8) M. DEX suppressed proliferation of BAL lymphocytes similarly at 4:00 P.M. and 4:00 A.M. in both groups. However, BAL macrophages from NA exhibited less suppression of IL-8 and TNF-alpha production by DEX at 4:00 A.M. as compared with 4:00 P.M. ($p < 0.0001$), whereas in the CNA group DEX suppressed IL-8 and TNF-alpha production equally at both time points. GRbeta expression was increased at night only in NA, primarily due to significantly increased expression by BAL macrophages ($p = 0.038$). IL-13 mRNA expression was increased at night in NA, but only in the NA group after addition of neutralizing ***antibodies*** to ***IL*** - ***13***, revealed GRbeta expression by BAL macrophages. We conclude that the airway macrophage may be the airway inflammatory cell driving the reduction in steroid responsiveness at night in NA, and this function is modulated by IL-13.

L28 ANSWER 6 OF 16 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 2002268868 MEDLINE
DOCUMENT NUMBER: 21955974 PubMed ID: 11795676
TITLE: Childhood asthma as an allergic disease revisited: implications for the development of future treatment
AUTHOR: Lang M L; Powell C V

[illegible]

L28 ANSWER 7 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
ACCESSION NUMBER: 2002:186429 BIOSIS
DOCUMENT NUMBER: PREV200200186429
TITLE: NFkappaB-activation in Hodgkin- Reed-Sternberg-cells can be decreased by inhibition of interleukin-13-signalling.
AUTHOR(S): Knorre, Alexander (1); Skonnier, Brian E.; Kaiser, Stefan; Schald, Walter; Pahl, Heike (1); Mak, Tak W.; Kapp, Ursula
CORPORATE SOURCE: (1) Experimental Anaesthesiology, University Medical Center, Freiburg Germany
SOURCE: Blood, (November 16, 2001) Vol. 98, No. 11, Part 1, pp. 305a, http: www.bloodjournal.org/; print.
Meeting Info.: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001
ISSN: 0006-4971.
DOCUMENT TYPE: Conference
LANGUAGE: English

AB The unique cellular background of reactive cells surrounding the rare population of Hodgkin- Reed-Sternberg (HRS) cells in Hodgkin specimen and the systemic clinical symptoms of Hodgkin lymphoma (HL) suggest that cytokines play a role in the pathogenesis of the disease. We have demonstrated previously that interleukin (IL)-13 is strongly expressed and secreted by some Hodgkin-derived cell lines and also expressed by HRS cells in primary tissue. Specific expression of IL-13 could be found in 25 to 100% of HRS cells in 86% of 80 cases of classical HL tested by in situ hybridisation. Furthermore we were able to demonstrate that proliferation of the IL-13 secreting cell lines HDLM2 and L1236 can be inhibited by treatment with IL-13 neutralizing antibodies. These findings suggest that an autocrine stimulation by IL-13 might be one step in the multistep transformation process of HL. Another pathway that might play a role for proliferation and survival of HRS cells is activation of NF-kappaB. As opposed to normal B cells constitutive presence of NF-kappaB1 relA could be demonstrated in the nucleus of HRS-cells. Here we investigate [illegible]

[illegible]

[illegible] IL-4 induced [illegible] GRbeta [illegible] IL-4 treatment cells were harvested and investigated for nuclear NF-kappaB by gel shift and [illegible]

proliferation. In HDLM2 cell neutralization of IL-13, as well as blockade of the IL-13 IL-4R leads to a significant loss of nuclear NF-kappaB1 relA. In L1236 NF-kappaB activation was not altered by IL-13 neutralization. This study indicates that NF-kappaB1 relA activation may be linked to IL-13 signalling mediated by the IL-13 IL-4R in HL-derived cells. Proliferation of the cell line L1236 can be inhibited by IL-13 neutralization without inactivation of NF-kappaB1 relA, which suggests that the proliferative effect of IL-13 on HL cells might not depend on NF-kappaB activation.

L28 ANSWER 8 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:129980 BIOSIS
DOCUMENT NUMBER: PREV200200129980
TITLE: Interleukin 13 (IL-13) levels in serum from patients with Hodgkin disease (HD) and healthy volunteers.
AUTHOR(S): Fumiara, Paolo (1); Cabanillas, Fernando (1); Younes, Anas (1)
CORPORATE SOURCE: (1) Lymphoma Myeloma, M.D. Anderson Cancer Center, Houston, TX USA
SOURCE: Blood, (November 16, 2001) Vol. 98, No. 11 Part 1, pp. 129a. http://www.bloodjournal.org/ print.
Meeting Info.: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001
ISSN: 0006-4971.
DOCUMENT TYPE: Conference
LANGUAGE: English
AB Interleukin 13 (IL-13) has recently been found to be highly expressed in cultured Hodgkin disease (HD)-derived cell lines and primary Hodgkin and Reed-Sternberg (H/RS) cells. Furthermore, IL-13 has been detected in the supernatants of HD-derived cell lines by enzyme-linked immunosorbent assay (ELISA), and neutralizing ***antibody*** to ***IL***-***13*** results in inhibition of H/RS cell proliferation in vitro. Because of the potential therapeutic implication of these observations, we examined IL-13 levels in serum from patients with newly diagnosed and relapsed HD and healthy volunteers. Supernatants from 3 HD-derived cell lines (HD-LM2, L-428, and KMH-2) known to produce IL-13 were used as positive controls. The sensitivity of the ELISA assay is less than 12 pg/ml. As previously reported, all 3 HD cell lines produced IL-13 (range 85-300 pg/ml). In contrast, IL-13 was below the detectable level in sera from 40 healthy individuals tested. Subsequently, we examined IL-13 levels in sera from 108 newly diagnosed patients with HD (70% had nodular sclerosis histology). Thirty one (28%) had B symptoms, and 36% had stage III/IV presentation. IL-13 levels were elevated in sera from 11 (10%) of 108 patients (range 34 to 82 pg/ml). However, IL-13 levels did not correlate with B symptoms, disease bulk, histologic subtype, advanced Ann Arbor stage, or shorter disease free survival. Of the 11 newly diagnosed patients who had elevated serum IL-13 levels, only one patient experienced disease progression, 4 months after completing therapy for stage IIB bulky disease. We also studied IL-13 levels in sera from 31 patients with relapsed HD. Five (16%) had elevated IL-13 serum levels (range 42 to 68 pg/ml). Our data show for the first time that IL-13 levels can be elevated in the serum of patients with HD. Although the number of patients with elevated IL-13 levels is small, this does not rule out the possibility of higher concentrations at the disease site. Our data may serve as the basis for new treatment strategies to explore the potential clinical relevance of IL-13 in patients with HD.

L28 ANSWER 9 OF 16 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2001-080753 [09] WPIDS
DOC. NO. CPI: C2001-023298
TITLE: Treating tissue fibrosis and or inhibiting formation of tissue fibrosis in a mammalian subject, involves administering a pharmaceutical composition comprising IL-13 antagonist

DK EE ES FI GB GE
GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG
MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG
UZ VN YU ZW
AU 2000057561-A 20020109 (200122)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 200078336-A1		WO 2000-US17102	20000621
AU 2000057561-A		AU 2000-57561	20000621

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000057561-A	Based on	WO 200078336

PRIORITY APPLN. INFO: US 1999-334512 19990621
AN 2001-080753 [09] WPIDS
AB WO 200078336-A UPAB: 20010213
NOVELTY - Treating tissue fibrosis and or inhibiting formation of tissue fibrosis in a mammalian subject, comprising administering a pharmaceutical composition (C1) comprising a protein (I), or a composition (C2) comprising a molecule (II) which is interleukin (IL) 13 or IL-4 antagonist, is new.
DETAILED DESCRIPTION - Treating tissue fibrosis and or inhibiting formation of tissue fibrosis in a mammalian subject, comprising administering a pharmaceutical composition (C1) comprising a protein (I), or a composition (C2) comprising a molecule (II) which is interleukin (IL)-13 or IL-4 antagonist, is new. (I) comprises a 383 residue amino acid sequence, (S1), fully defined in the specification, residues 22-334 or 357-383 of S1, a 380 residue amino acid sequence (S2), fully defined in the specification, amino acids 26-341 or 363-380 of S2, or fragments of S1 or S2 having a biological activity of IL-13 receptor binding chain.
ACTIVITY - Cytostatic.
MECHANISM OF ACTION - Inhibitor of tissue fibrosis formation (claimed).
C57BL/6 WT and IL-4-deficient mice were infected percutaneously with 25 Schistosoma mansoni cercariae. Separate groups of animals were treated with either sIL-13R alpha 2-Fc or with control-Fc. The treatments began on week 5, at the start of egg laying, and all animals were sacrificed 8 week post-infection and examined for several parasitologic and immunologic parameters. All four groups of mice harbored similar worm burdens, and tissue eggs produced per worm pair did not vary among the groups. At 8 week post-infection, the time of peak tissue response 45, WT mice showed no significant change in granuloma size as a result of IL-13 blockade. Control-Fc-treated IL-4-deficient mice also failed to show a reduced granulomatous response, and in fact, granulomas were significantly larger in these mice. In striking contrast to these observations, the IL-4-deficient mice displayed a markedly reduced granulomatous response when IL-13 was inhibited. The double IL-4-deficient sIL-13R alpha 2-Fc-treated mice displayed on average a 40-50% reduction in granuloma volume when compared with either control or sIL-13R alpha 2-Fc-treated WT animals, and more than a 75% reduction when compared with control-Fc-treated IL-4 deficient mice. The sIL-13R alpha 2 treatment significantly reduced the collagen content of liver granulomas in WT mice and decreased liver hydroxyproline levels, while the IL-4 deficiency related in a less significant reduction. The overall results showed that treatment with sIL-13R alpha 2-Fc significantly reduced hepatic fibrosis in S. mansoni-infected mice.
MECHANISM OF ACTION - IL-13 or IL-4 inhibitor antagonist.
USE - The method is useful for treating or inhibiting the formation of tissue fibrosis resulting from infection with schistosoma or from healing of a surgical incision wound. Fibrosis affects skin epidermis, skin endodermis, muscle, tendon, cartilage, tissues of cardiac, pancreatic, lung, uterine, neural, testis, ovary, adrenal gland, artery, vein, colon, small intestine, biliary tract or gut tissue or more preferably liver tissue (claimed).
Dwg.0/7

L28 ANSWER 10 OF 16 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2001-024676 [03] WPIDS
DOC. NO. CPI: C2001-007458

DK EE ES FI GB GE
GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG
MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG
UZ VN YU ZW
AU 2000046805-A 20001116 (200109)
EP 1173484-A1 20020123 (200214) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT
RO SI
CN 1348465-A 20020508 (200253)
HU 2002000862-A2 20020729 (200258)
KR 2002026426-A 20020410 (200267)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 200064944-A1		WO 2000-US11612	20000428
AU 2000046805-A		AU 2000-46805	20000428
EP 1173484-A1		EP 2000-928591	20000428
		WO 2000-US11612	20000428
CN 1348465-A		CN 2000-806772	20000428
HU 2002000862-A2		WO 2000-US11612	20000428
		HU 2002-862	20000428
KR 2002026426-A		KR 2001-713820	20011029

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000046805-A	Based on	WO 200064944
EP 1173484-A1	Based on	WO 200064944
HU 2002000862-A2	Based on	WO 200064944

PRIORITY APPLN. INFO: US 1999-301808 19990428
AN 2001-024676 [03] WPIDS
AB WO 200064944-A UPAB: 20001116
NOVELTY - Treating or inhibiting formation of tissue fibrosis in a mammalian subject, comprises administering a composition comprising an interleukin(IL) 13 antagonist or an IL-4 antagonist.
ACTIVITY - Vulnerary. The effect of IL-13 inhibitor antagonist such as soluble IL-13R alpha 2-Fc in preventing fibrosis associated with chronic infectious diseases was studied. C57BL/6 WT and IL-4 deficient mice were infected percutaneously with Schistosoma mansoni cercariae. Separate groups of animals were treated with either sIL-13R alpha 2-Fc or with control-Fc. All animals were sacrificed 8 week postinfection and examined for several parasitologic and immunologic parameters. WT mice showed a significant change in granuloma size as a result of IL-13 blockade. Control Fc-treated IL-4 deficient mice also failed to show a reduced granulomatous response. The IL-4 deficient mice displayed a markedly reduced granulomatous response when IL-13 was inhibited. The double IL-4-deficient sIL-13R alpha 2-Fc-treated mice displayed a 40-50% reduction in granuloma volume when compared with either control or sIL-13R alpha 2-Fc treated WT animals, and more than a 75% reduction when compared with control-Fc-treated IL-4 deficient mice. The sIL-13R alpha 2 treatment significantly reduced the collagen content of liver granulomas in WT mice and decreased liver hydroxyproline levels, while the IL-4 deficiency related in a less significant reduction. The overall results showed that treatment with sIL-13R alpha 2-Fc significantly reduced hepatic fibrosis in S. mansoni-infected mice.
MECHANISM OF ACTION - IL-13 or IL-4 inhibitor antagonist.
USE - The method is useful for treating or inhibiting the formation of tissue fibrosis resulting from infection with schistosoma or from healing of a surgical incision wound. Fibrosis affects skin epidermis, skin endodermis, muscle, tendon, cartilage, tissues of cardiac, pancreatic, lung, uterine, neural, testis, ovary, adrenal gland, artery, vein, colon, small intestine, biliary tract or gut tissue or more preferably liver tissue (claimed).
Dwg.0/7

L28 ANSWER 11 OF 16 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2001-433587 [37] WPIDS

WO 2000036103 A1 20000622 (200037)* EN 60
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
W: AE AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZW
AU 2000021775 A 20000703 (200046)
EP 1141286 A1 20011010 (200167) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
BR 9916209 A 20011226 (200206)
CN 1352686 A 20020605 (200261)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000036103	A1	WO 1999-US29493	19991213
AU 2000021775	A	AU 2000-21775	19991213
EP 1141286	A1	EP 1999-966166	19991213
		WO 1999-US29493	19991213
BR 9916209	A	BR 1999-16209	19991213
		WO 1999-US29493	19991213
CN 1352686	A	CN 1999-815591	19991213

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000021775	A	Based on	WO 200036103
EP 1141286	A1	Based on	WO 200036103
BR 9916209	A	Based on	WO 200036103

PRIORITY APPLN. INFO: US 1998-211335 19981214
AN 2000-431587 [37] WPIDS
AB WO 200036103 A UPAB: 20000807
NOVELTY - A polynucleotide comprising a nucleotide sequence that encodes an interleukin-13 binding chain (IL-13bc) or fragment, of IL-13 receptor, is new.
DETAILED DESCRIPTION - The polynucleotide comprises a nucleotide sequence that is:
(a) nucleotides 256 to 1404 of a 1525 murine nucleotide sequence, given in the specification;
(b) nucleotides 103 to 1242 of a 1369 human nucleotide sequence, given in the specification;
(c) a variant of (a) or (b) as a result of degeneracy of the genetic code;
(d) hybridizable under stringent conditions to (a) or (b);
(e) a species homolog of (a) or (b); or
(f) an allelic variant of (a) or (b).
INDEPENDENT CLAIMS are also included for the following:
(1) a host cell transformed with the new polypeptide;
(2) producing an IL-13bc (binding chain) protein comprising growing a culture of the host cell in culture medium and purifying IL-13bc from the culture;
(3) an isolated IL-13bc protein comprising a sequence of
(I) 383 amino acids, given in the specification;
(II) amino acids 22 to 334 of (I);
(III) amino acids 357 to 383 of (I);
(IV) 380 amino acids, given in the specification;
(V) amino acids 26 to 341 of (IV);
(VI) amino acids 363 to 380 of (IV), or
(VII) fragments of (I) to (VI) having IL-13 receptor binding chain activity
(4) a protein produced by (2);
(5) a composition comprising an antibody that reacts with (3);
(6) identifying an inhibitor of IL-13 binding to the IL-13 receptor (IL-13R) comprising:
(i) combining (2) with IL-13 or a fragment to form a first binding mixture;
(ii) measuring binding between the protein and IL-13 or fragment;
(iii) combining a compound with the protein and IL-13 or fragment to form a second binding mixture;
(iv) measuring the amount of binding; and
(v) comparing the binding in the first binding mixture with the

(12) inhibiting interaction of IL-13 with IL-13bc in a mammal by administering IL-13 antagonist
ACTIVITY - Antiallergic; antiinflammatory; antiasthmatic; dermatological; immunosuppressive; antithyroid; cytostatic
Male A/J mice were immunized intraperitoneally and challenged intratracheally with soluble ovalbumin. The allergic phenotype was assessed 4 days after the antigen challenge. Blockade of IL-13 was performed 24 hours before the allergen challenge by systemic administration of soluble IL-13bc-IgGFc fusion protein which binds to and neutralizes IL-13. Challenge of allergen-immunized mice resulted in significant increases in airway responsiveness to acetylcholine. Blockade of IL-13 resulted in complete reversal of the established allergen-induced airway hyper responsiveness, showing that asthma may be treated.
MECHANISM OF ACTION - IL-13 inhibitor.
USE - For identifying and producing an IL-13bc protein that can inhibit the binding of IL-13 to an IL-13 receptor and treat an IL-13-related condition such as an IgE-mediated condition. Atopy, allergic conditions, asthma, immune complex diseases, lupus, nephritis, thyroiditis, Grave's disease or inflammatory conditions of the lung can be treated. For potentiating IL-13 activity (all claimed). Cancer may be treated. Macrophage activation is enhanced allowing use in vaccination and treatment of mycobacterial, intracellular organisms, or parasitic infections.
Dwg.0/4

L28 ANSWER 12 OF 16 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1999307279 MEDLINE
DOCUMENT NUMBER: 99307279 PubMed ID: 10377189
TITLE: Interleukin 13 is secreted by and stimulates the growth of Hodgkin and Reed-Sternberg cells.
AUTHOR: Kapp U; Yeh W C; Patterson B; Elia A J; Kagi D; Ho A; Hessel A; Tipsword M; Williams A; Mirtsos C; Itie A; Moyle M; Mak T W
CORPORATE SOURCE: Amgen Institute, Ontario Cancer Institute, the Department of Medical Biophysics, and the Department of Immunology, University of Toronto, Toronto, Ontario M5G 2C1, Canada.
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1999 Jun 21) 189 (12) 1939-46.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990806
Last Updated on STN: 19990806
Entered Medline: 19990726
AB Gene expression patterns can provide vital clues to the pathogenesis of neoplastic diseases. We investigated the expression of 950 genes in Hodgkin's disease (HD) by analyzing differential mRNA expression using microarrays. In two independent microarray experiments, the HD-derived cell lines L428 and KMH2 were compared with an Epstein-Barr virus (EBV)-immortalized lymphoblastoid B cell line, LCL-GK. Interleukin (IL)-13 and IL-5 were found to be highly expressed in the HD-derived cell lines. Examination of IL-13 and IL-5 expression by Northern blot analysis and enzyme-linked immunosorbent assay confirmed these results and revealed the expression of IL-13 in a third HD-derived cell line, HDLM2. Control LCL and EBV-negative non-Hodgkin lymphoma-derived cell lines did not express IL-13. In situ hybridization of lymph node tissue from HD patients showed that elevated levels of IL-13 were specifically expressed by Hodgkin/Reed-Sternberg (H/RS) tumor cells. Treatment of a HD-derived cell line with a neutralizing ***antibody*** to ***IL*** - ***13*** resulted in a dose-dependent inhibition of H/RS cell proliferation. These data suggest that H/RS cells produce IL-13 and that IL-13 plays an important role in the stimulation of H/RS cell growth, possibly by an autocrine mechanism. Modulation of the IL-13 signaling pathway may be a logical objective for future therapeutic strategies.

L28 ANSWER 13 OF 16 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 1999335604 MEDLINE
DOCUMENT NUMBER: 99335604 PubMed ID: 10404009

ENTRY DATE: Entered STN: 19991012
Last Updated on STN: 19991012
Entered Medline: 19990928
AB Rheumatoid arthritis (RA) is an autoimmune disease characterized by a heavy lymphocytic infiltration into the synovial cavity, resulting in the secretion of a variety of cytokines which ultimately leads to destruction of joint tissue. Among the infiltrating cells are activated T cells which produce specific cytokines capable of osteoclast progenitor cell expansion, fusion, and activation. Cultures of activated human T cells and human osteoblasts (hOBs) were used to study the possibility that lymphokines may act on osteoblasts to produce the osteoclastogenic factor interleukin-6 (IL-6). Purified T cells were activated with a combination of anti-CD3 and anti-CD28 antibodies, cocultured with hOBs in direct physical contact or separated by a transwell system, and conditioned media (CM) were assayed for IL-6 production. After a 72 h incubation period, activated T cell-hOB interaction resulted in a 100-fold increase of IL-6 production over basal levels. The immunosuppressant cyclosporine A (CsA) inhibited T cell tumor necrosis factor alpha and IL-6 production but did not inhibit the T cell induction of IL-6 from hOB. Assay of activated T-cell CM on hOB revealed that a soluble factor, not cell-cell contact, was the major inducer of IL-6. The induction of IL-6 mRNA by both activated T cell CM and CsA-treated activated T cell CM was confirmed by Northern blot analysis. Neutralizing ***antibodies*** to ***IL*** - ***13*** and IL-17 did not affect IL-6 production. These findings suggest that activated T cells produce a novel, potent, IL-6-inducing factor that may be responsible for the bone loss observed in RA patients.

L28 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:433168 CAPLUS
DOCUMENT NUMBER: 121:33168
TITLE: Human interleukin-13 and the gene encoding it
INVENTOR(S): Aversa, Gregorio; Banchereau, Jacques; Briere, Francine; Cocks, Benjamin G.; Coffman, Robert L.; Culpepper, Janice; Dang, Warren; De Vries, Jan; De Waal, Malefyt Rene; et al.
PATENT ASSIGNEE(S): Schering Corp., USA
SOURCE: PCT Int. Appl., 136 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9404680	A1	19940303	WO 1993-US7645	19930818
W: AU, BB, BG, BR, BY, CA, CZ, FI, HU, JP, KR, KZ, LK, MG, MN, MW, NO, NZ, PL, RO, RU, SD, SK, UA, VN				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5596072	A	19970121	US 1993-12543	19930201
EP 656947	A1	19950614	EP 1993-920049	19930818
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 07508179	T2	19950914	JP 1993-506436	19930818

PRIORITY APPLN. INFO.: US 1992-933416 19920821
US 1993-10977 19930129
US 1993-12543 19930201
WO 1993-US7645 19930818
AB A cDNA encoding human interleukin 13 (IL-13) is cloned and expressed and the immunol. properties of the protein characterized. Polyclonal and monoclonal antibodies to the protein are prepd. and methods of using the cDNA and protein in diagnostics and therapeutics are described. A cDNA for the protein was cloned from a T cell cDNA libraries by repeated screening with a cDNA for mouse P600 protein to obtain overlapping clones from which a full-length cDNA was constructed. The protein was manufd. as a fusion protein with glutathione-S-transferase and purified from inclusion bodies by solubilization, refolding, and cleavage with thrombin. Human IL-13 stimulated B-cell DNA synthesis through the antigen receptor and acted as a growth factor for B cells stimulated through the CD40 antigen. IL-13 also stimulated IgE secretion in anti-CD40 activated B

Journal code: 1273201. ISSN: 0014-2980.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199407
ENTRY DATE: Entered STN: 19940721
Last Updated on STN: 19990129
Entered Medline: 19940708
AB Interleukin (IL)-13 is a newly described cytokine expressed by activated
lymphocytes. We examined the effects of the murine recombinant cytokine on
the phenotype and activation status of elicited peritoneal macrophages (M
phi), concentrating on activities which are known to be modulated by
interferon-gamma and IL-4. IL-13 markedly suppressed nitric oxide release
and to a lesser extent secretion of the pro-inflammatory cytokine tumor
necrosis factor-alpha. However, antimicrobial capacity was not completely
jeopardized as the respiratory burst was unaffected, and indeed the
enhanced expression of M phi mannose receptor and major histocompatibility
class II, and regulation of sialoadhesin, the M phi sialic acid-specific
receptor involved in hemopoietic and lymphoid interactions, suggest that
these cells are not simply deactivated, but primed for an active role in
immune and inflammatory responses. These activities closely mimic those of
IL-4, but mediation of the effects by IL-4 was discounted by the use of a
neutralizing monoclonal ***antibody***. Thus, ***IL*** -
13
, like IL-4, is a cytokine which has complex effects on M phi behavior,
inducing activities characteristic of both activation and deactivation.

L28 ANSWER 16 OF 16 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 95137668 MEDLINE
DOCUMENT NUMBER: 95137668 PubMed ID: 7530690
TITLE: IL-13 has only a subset of IL-4-like activities on B
chronic lymphocytic leukaemia cells
AUTHOR: Fluckiger A C; Briere F; Zurawski G; Bridon J M;
Banchereau
J
CORPORATE SOURCE: Schering-Plough, Laboratory for Immunological Research,
Dardilly, France.
SOURCE: IMMUNOLOGY, (1994 Nov) 83 (3) 397-403.
Journal code: 0374672. ISSN: 0019-2805.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199503
ENTRY DATE: Entered STN: 19950314
Last Updated on STN: 19960129
Entered Medline: 19950302
AB The recently described interleukin-13 (IL-13) has been shown to share many
of the effects of IL-4 on normal B cells, including growth-promoting
activity and induction of CD23. In this study we compared the effects of
IL-13 and IL-4 on B chronic lymphocytic leukaemias (B-CLL) cells. After
anti-CD40 activation, both IL-13 and IL-4 promoted the DNA synthesis of
B-CLL cells and increased the recovery of viable cells. The time kinetics
of the proliferative response of B-CLL cells to IL-13 or IL-4 were
superimposable and showed the long-lasting effect of both cytokines. As on
normal B cells, both IL-4 and IL-13 synergize with IL-10 to enhance B-CLL
DNA synthesis. Moreover, IL-13, like IL-4, was able to increase CD23
expression on anti-CD40-activated leukaemic B cells. The CD23
up-regulation and the DNA synthesis induced by IL-13 or
anti-CD40-activated B-CLL cells were significantly reduced when B-CLL
cells were cultured with anti IL-4 receptor monoclonal antibody,
suggesting a common pathway for IL-13 and IL-4 signalling. However, after
cross-linking of surface IgM, IL-4 strongly inhibited the IL-2-induced DNA
synthesis of B-CLL cells, whereas IL-13 did not inhibit IL-2-driven
proliferation of anti-IgM-activated B-CLL cells. Furthermore, while IL-4
strongly up-regulated the expression of CD23 on anti-IgM-activated
leukaemic B cells, IL-13 only marginally increased it. Finally, IL-13, in

FILE 'MEDLINE, JAPIO, BIOSIS, SCISEARCH, WPIDS, CAPLUS, EMBASE' ENTERED
AT 20:18:20 ON 19 OCT 2002
L1 10876 S IL-13 OR IL 13 OR INTERLEUKIN 13 OR INTERLEUKIN-13
L2 2282 L1 AND ANTIBOD?
L3 0 S IL-13 AND ANTIBOD?
L4 0 S IL 13 AND ANTIBOD?
L5 0 S INTERLEUKIN 13 AND ANTIBOD?
L6 0 S IL-13 AND ANTIBOD?
L7 0 S IL 13 AND ANTIBOD?
L8 0 INTERLEUKIN 13 AND ANTIBOD?
L9 0 INTERLEUKIN-13 AND ANTIBOD?
L10 0 INTERLEUKIN 13 AND ANTIBOD?
L11 0 INTERLEUKIN? WITH ANTIBOD?
L12 2098 INTERLEUKIN? WITH ANTIBOD?
L13 0 INTERLEUKIN?2 WITH ANTIBOD?
L14 0 S INTERLEUKIN13 ANTIBOD?
L15 0 S IL13 ANTIBOD?
L16 0 S IL13 ANTIBODIES
L17 0 S IL13 ANTIBODY
L18 46 IL-13 ANTIBODY
L19 21 ANTIBODIES TO IL-13
L20 41 ANTIBODY TO IL-13
L21 41 S ANTIBODY TO IL-13
L22 12 S ANTIBODY TO INTERLEUKIN 13
L23 11 S ANTIBODIES TO INTERLEUKIN 13
L24 11 DUP REM L18 (35 DUPLICATES REMOVED)
L25 7 DUP REM L19 (14 DUPLICATES REMOVED)
L26 16 DUP REM L20 (25 DUPLICATES REMOVED)
L27 12 DUP REM L22 (0 DUPLICATES REMOVED)
L28 16 DUP REM L24 (25 DUPLICATES REMOVED)
L29 11 DUP REM L25 (0 DUPLICATES REMOVED)

=d l27 ibib abs 1-12

L27 ANSWER 1 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:587646 CAPLUS
DOCUMENT NUMBER: 137:139373
TITLE: Compositions and methods for specifically targeting tumors
INVENTOR(S): Debinski, Waldemar; Puri, Raj K.
PATENT ASSIGNEE(S): Penn State University, USA
SOURCE: U.S., 38 pp., Cont.-in-part of U.S. 5,614,191.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6428788	B1	20020806	US 1996-706207	19960830
US 5614191	A	19970325	US 1995-404685	19950315
CA 2215122	AA	19960926	CA 1996-2215122	19960315
US 5919456	A	19990706	US 1997-821840	19970321
WO 9808957	A1	19980305	WO 1997-US15050	19970827

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
AU 9747340 A 19980319 AU 1997-47340 19970827
US 2002031492 A1 20020314 US 2001-894699 20010628
PRIORITY APPLN. INFO.: US 1995-404685 A2 19950315
US 1996-706207 A 19960830
WO 1997-US15050 W 19970827
US 1999-226794 A2 19990107
US 2000-215623P P 20000630

AB The present invention provides a method and compns. for specifically delivering an effector mol. to a tumor cell. The method involves providing a chimeric mol. comprising an effector mol. (such as Pseudemonas exotoxin, Diphtheria toxin, ricin, abrin, or cytotoxic drugs) attached to a targeting mol. (such as interleukin-13 or anti-interleukin-13 antibody) that specifically binds an interleukin-13 receptor and contacting a tumor cell with the chimeric mol. in the presence of an interleukin-4 receptor blocker (e.g. mutated interleukin-4).
REFERENCE COUNT: 1 THERE ARE 1 CITED

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001062933	A2	20010830	WO 2001-GB707	20010220
WO 2001062933	A3	20011220		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: GB 2000-4016 A 20000222

AB This invention relates to variants of the nucleic acid sequence encoding Interleukin 13 (IL-13) and the use of such sequence variants in medicine, esp. in the diagnosis of susceptibility or resistance to disorders assocd. with an immune response, particularly the inflammatory response assocd. with asthma, atopic allergy and latex sensitivity. Unexpectedly, by comparing the IL-13 gene sequences deposited in the GenBank™ database, upstream of nucleotide +80, we identified four single nucleotide variations in four of the deposited sequences of the IL-13 gene. The four potential single nucleotide polymorphisms (SNP's) were: a G/C at +543nt, a C/T at +1922nt, a G/A at +2043nt and a C/A at +2579nt upstream of the first nucleotide of the start codon (figure 1; [SEQ ID No 1]), which represent nucleotide positions 1314, 2693, 2814 and 3350 resp. in GenBank™ deposited sequence L13029. Moreover, the G to A substitution at position +2043nt was found to change the codon sequence CGC that codes for the basic amino acid arginine (Arg) at amino acid position 130 of the unprocessed precursor (see GenBank™ deposited sequence P35225), to CAG that codes for the hydrophilic amino acid glutamine (Gln) (see figure 2; [SEQ ID No 2]). The invention also provides a transgenic, nonhuman mammalian animal whose germ cells and somatic cells contain a nucleic acid mol. The invention further provides the use of an amino acid sequence in a method of producing an antibody, for use in detecting susceptibility or resistance to a disorder assocd. with an immune response.

L27 ANSWER 3 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:535020 CAPLUS
DOCUMENT NUMBER: 133:149151
TITLE: Materials and methods to inhibit Hodgkin and Reed Sternberg cell growth
INVENTOR(S): Mak, Tak W.; Kapp, Ursula
PATENT ASSIGNEE(S): Amgen Canada, Can
SOURCE: PCT Int. Appl., 28 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000044407	A2	20000803	WO 2000-CA94	20000201

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE,

components and IL-13 assocd. signal transduction pathways. Methods for detecting IL-13 receptor components in the body fluids, and methods for the inhibitors to treat Hodgkin's disease are also disclosed.

TITLE: Method for diagnosing, imaging, and treating tumors bearing interleukin 13-specific receptors
INVENTOR(S): Debinski, Waldemar; Connor, James R
PATENT ASSIGNEE(S): The Penn State Research Foundation, USA
SOURCE: PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000040264	A1	20000713	WO 2000-US149	20000105

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU,
CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL,
IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA,
MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI,
SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
US 2001053371 A1 20011220 US 1999-226794 19990107
EP 1140167 A1 20011010 EP 2000-901390 20000105
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO
JP 2002534395 T2 20021015 JP 2000-592019 20000105
PRIORITY APPLN. INFO.: US 1999-226794 A 19990107
WO 2000-US149 W 20000105

AB Disclosed is a method of inhibiting the growth of tumors bearing IL-13-specific receptors. Included among this class of tumors is glioblastoma multiforme (GBM), a rapidly progressing brain tumor for which there is currently no effective treatment available. In the disclosed method, a chimeric cytotoxin comprising an IL-13 receptor-binding moiety and a cytotoxic moiety is delivered into a mammalian subject having a tumor bearing IL-13-specific receptors. All studied human GBM specimens abundantly express the IL-13-specific tumor.
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L27 ANSWER 5 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:180729 CAPLUS
DOCUMENT NUMBER: 128:256388
TITLE: Therapeutic molecules
INVENTOR(S): Nicola, Nicos Antony; Hilton, Douglas James; Zhang, Jian-Guo; Simpson, Richard John
PATENT ASSIGNEE(S): Amrad Operations Pty. Ltd., Australia; Nicola, Nicos Antony; Hilton, Douglas James; Zhang, Jian-Guo; Simpson, Richard John
SOURCE: PCT Int. Appl., 70 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9813063	A1	19980402	WO 1997-AU641	19970930

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR,
KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ,
PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG,
US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR,
GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA,

IL-13BP
mutant gene

L27 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:288048 CAPLUS
DOCUMENT NUMBER: 129:26848
TITLE: Regulation of interleukin-13 receptor constituents on mature human B lymphocytes
AUTHOR(S): Ogata, Haruki; Ford, Dwayne; Kouttab, Nicola; King, Thomas C.; Vita, Natalio; Minty, Adrian; Stoeckler, Johanna; Morgan, Deborah; Girasole, Christopher; Morgan, John W.; Marzel, Abby I.
CORPORATE SOURCE: Roger Williams Med. Cent., Brown Univ., Providence, RI, 02908, USA
SOURCE: Journal of Biological Chemistry (1998), 273(16), 9864-9871
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Human B cells stimulated through both their Ig and CD40 receptors up-regulate 745 interleukin (IL)-13 ligand binding sites with an affinity of 0.91 nM within 24 h. IL-13 binds primarily to the IL-13R.alpha.1 with subsequent sequestration of the IL-4R.alpha. into the complex. IL-13R.alpha.1 may also be found in those receptors capable of binding IL-4: .gamma.c Chain (.gamma.c) participates in receptors capable of binding IL-4 but is not found in assocn. with bound IL-13. Dimeric receptors composed of the IL-4R.alpha. complexed with either the IL-13R.alpha.1 or .gamma.c occur simultaneously within defined B cell populations. mRNAs for all receptor constituents are increased subsequent to Ig stimulation alone, while maximal expression of IL-13R.alpha.1 is more dependent upon co-stimulation of Ig and CD40 receptors. MRNA levels for IL-13R.alpha.1 vary over a wider range subsequent to surface stimulation than other receptor components. Although .gamma.c is not bound to IL-13 in B cells under the conditions evaluated, it may influence IL-13 binding by competing with IL-13R.alpha.1 for assocn. sequestration with the IL-4R.alpha. chain. IL-13R.alpha.2 does not participate in the IL-13 receptor that is up-regulated upon activation of quiescent tonsillar B lymphocytes, although mRNA for the protein may be found in the centroblastic fraction of tonsillar cells.

L27 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:594756 CAPLUS
DOCUMENT NUMBER: 127:258660
TITLE: Cloning and expression of cDNA for interleukin-13 binding chain of IL-13 receptor, identification of inhibitors of binding, and treatment of Ig-mediated disease
INVENTOR(S): Collins, Mary; Donaldson, Debra; Fitz, Lee; Neben, Tamlyn; Whitters, Matthew; Wood, Clive
PATENT ASSIGNEE(S): Genetics Institute, Inc., USA
SOURCE: PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9733946	A1	19970918	WO 1997-US3124	19970228

W: AU, CA, JP, MX
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
US 5710023 A 19980120 US 1996-610572 19960301
AU 9725980 A1 19970916 AU 1997-25980 19970228
US 6214339 B1 20010410 US 1997-841781 19970430
US 6248574 B1 20010619 US 1997-846340 19970430
US 6268480 B1 20010731 US 1997-846344 19970430
PRIORITY APPLN. INFO.: US 1996-610572 A 19960301
WO 1997-US3124 W 19970228

AB Polynucleotides encoding the IL-13-binding subunit of the IL-13 receptor and fragments thereof are disclosed. IL-13 receptor proteins, methods for their prodn., inhibitors of binding of IL-13 and its receptor and methods for their identification are also disclosed. Use of the inhibitors for treatment of Ig-mediated diseases such as allergies, asthma, lupus, etc.

Laurent,
Patrick; Vita, Natalio
SOURCE: PCT Int. Appl., 82 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9729126	A1	19970812	WO 1996-FR1756	19961107

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT,
RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN,
AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR,
IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML,
MR, NE, SN, TD, TG
FR 2742156 A1 19970613 FR 1995-14424 19951206
CA 2238893 AA 19970612 CA 1996-2238893 19961107
AU 9675760 A1 19970627 AU 1996-75760 19961107
EP 876482 A1 19981111 EP 1996-938273 19961107
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, FI
BR 9611697 A 19990217 BR 1996-11697 19961107
JP 11511028 T2 19990928 JP 1996-521017 19961107
ZA 9610238 A 19980605 ZA 1996-10238 19961205
NO 9802550 A 19980605 NO 1998-2550 19980605
PRIORITY APPLN. INFO.: FR 1995-14424 A 19951206
WO 1996-FR1756 W 19961107

AB Human interleukin 13 (IL-13) receptors are identified and cDNAs encoding them are cloned for diagnostic and therapeutic use. Two subunits of the receptor are described: one (IL-13.alpha.) is specific for IL-13 and the other (IL-13.beta.) is involved in the binding of IL-13 to the interleukin 4 receptor. The receptors can be used to increase the effectiveness of IL-13 by increasing the level of the receptor, or inhibiting IL-13, e.g. with antibodies to the receptor or a sol. form of the receptor. The cDNAs can be used to detect mutant alleles of the genes for the subunits in the diagnosis of immune disorders (no data). Mouse cDNAs for the receptors were used to design primers and probes for the cloning of the human receptors. A sol. form of one of the subunits was capable of antagonizing IL-13. The receptor was involved in the activation of the transcription factor STAT6.

L27 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:425227 CAPLUS
DOCUMENT NUMBER: 127:30144
TITLE: Interleukin-13 receptor .alpha.-chain protein NF4, mouse and human cDNA sequences, and applications in assays for asthma and allergy therapeutics and diagnostics
INVENTOR(S): Wilson, Tracy; Nicola, Nicos A.; Hilton, Douglas J.; Metcalf, Donald; Zhang, Jian Guo
PATENT ASSIGNEE(S): Amrad Operations Pty. Ltd., Australia; Wilson, Tracy; Nicola, Nicos A.; Hilton, Douglas J.; Metcalf, Donald; Zhang, Jian Guo
SOURCE: PCT Int. Appl., 92 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9715663	A1	19970501	WO 1996-AU668	19961023

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT,
RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US,

AB Polynucleotides encoding the IL-13 receptor .alpha.-chain protein NF4, with a greater affinity than the interleukin 13 receptor .alpha. chain (IL-13R.alpha.) and having mol. wt. about 46 kDa. The

L27 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:[illegible]
DOCUMENT NUMBER: 127:[illegible]
TITLE: Interleukin-13 receptor subunits of human, cDNAs encoding them, and their diagnostic and therapeutic

L27 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:[illegible]
DOCUMENT NUMBER: 127:[illegible]
TITLE: [illegible]
INVENTOR(S): [illegible]
PATENT ASSIGNEE(S): [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

[illegible]
AB [illegible]

L27 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:[illegible]
DOCUMENT NUMBER: 127:[illegible]
TITLE: [illegible]
INVENTOR(S): [illegible]
PATENT ASSIGNEE(S): [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

[illegible]
AB [illegible]

AU 1996-2208 A 19960909
WO 1996-AU668 W 19961023
US 1998-51843 A1 19980629

AB The present invention relates generally to a novel hematopoietin receptor,
NR4, which is the interleukin-13 receptor alpha-chain, or components or
parts thereof and to genetic sequences encoding the same. The receptor
mols. and their components and or parts and the genetic sequences encoding
same of the present invention are useful in the development of a wide
range of agonists, antagonists, therapeutics and diagnostic reagents based
on ligand interaction with its receptor. Mouse and human NR4 cDNA
sequences are included.

L27 ANSWER 3 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:708428 CAPLUS
DOCUMENT NUMBER: 125:317337
TITLE: Interleukin-13 receptor-specific chimeric proteins and their uses to treat tumors
INVENTOR(S): Puri, Raj K.; Debinski, Waldemar; Pastan, Ira; Obiri, Nicholas
PATENT ASSIGNEE(S): The Government of the United States of America, USA
SOURCE: PCT Int. Appl., 76 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9607417	A1	19960926	WO 1996-US3486	19960315

W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN
US 5614191 A 19970325 US 1995-404685 19950315
CA 2215122 AA 19960926 CA 1996-2215122 19960315
AU 9653110 A1 19961008 AU 1996-53110 19960315
AU 714541 B2 20000106
EP 1007696 A1 20000614 EP 1996-909693 19960315
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
JP 2000511042 T2 20000829 JP 1996-528499 19960315
US 5919456 A 19990706 US 1997-821840 19970321
PRIORITY APPLN. INFO.: US 1995-404685 A 19950315
WO 1996-US3486 W 19960315

AB A method and compns. are provided for specifically delivering an effector
mol. to a tumor cell. The method involves providing a chimeric mol. that
comprises an effector mol. attached to a targeting mol. that specifically
binds an interleukin-13 (IL-13) receptor and contacting a tumor cell with
the chimeric mol. The target moiety of the the chimeric mol. may consist
of IL-13, an anti-IL-13 receptor antibody, or circularly permuted IL-13.
the effector moiety may be a cytotoxin (Pseudomonas exotoxin, Diphtheria
toxin, ricin, or abrin), label, radionuclide, drug, liposome, ligand, or
antibody. Thus, recombinant DNA technol. was used to produce single-chain
fusion proteins human IL-13 (or its circularly permuted analog) to either
of 2 mutant forms of Pseudomonas aeruginosa exotoxin A. One variant
protein of IL-13 is a deriv. in which the normal N- and C-termini are linked
via the Gly-Gly-Ser-Gly linker peptide, and the bond between Gly-43 and
Met-44 is broken, thereby yielding cpIL-13 in which Met-44 is the new
N-terminus and Gly-43 is the new C-terminus. PE38QQR is a truncated form
of Pseudomonas exotoxin composed of amino acids 253-364 and 381-608, the
lysine residues at positions 590 and 606 are replaced by Gln and at 613 is
replaced by Arg. P38IL is a full-length Pseudomonas exotoxin with a

L27 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:352585 CAPLUS
DOCUMENT NUMBER: 125:31750
TITLE: IL-13 released by and localized in human basophils
AUTHOR(S): Lu, Huamin; Sim, Tommy C.; Alam, Reteul
CORPORATE SOURCE: Department Internal Medicine, University Texas Medical Branch, Galveston, TX, 77555, USA
SOURCE: Journal of Immunology (1996), 156(12), 4833-4838
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We and others have shown that human basophils can synthesize and release
IL-4. However, IL-13, a cytokine that closely resembles IL-4, has not
hitherto been described as a basophil product. The prodn. of IL-13 by
basophils was demonstrated by immunocytochem. Approx. 70% of basophils
stimulated with anti-Fc epsilon RI alpha. (antibody to the .alpha. subunit
of IgE receptor type I) stained for IL-13. Under similar exptl.
conditions, mononuclear cells failed to stain for IL-13. The cytokine was
localized to basophilic granules by electron microscopic examn. of
immunogold staining. The secretion of IL-13 into the culture supernatant
was assayed by ELISA. Kinetic studies showed detectable IL-13 release at
3 h, which steadily increased up to 24 h. This is significantly different
from the kinetics of basophil histamine and IL-4 release. IL-13 prodn.
was also obsd. upon stimulation with anti-IgE, anti-Fc epsilon RI alpha.,
IL-3, and A23187 in a dose-dependent manner. PBMC, neutrophils, and
eosinophils isolated from the same donors did not release IL-13 after
anti-IgE stimulation. The anti-IgE-induced basophil IL-13 synthesis could
be enhanced by IL-3 preincubation (with and without IL-3 preincubation,
anti-IgE-induced IL-13 prodn. was 227 and 42 pg/10^6 basophils, resp.).
PBMC produced a significant amt. of IL-13 upon stimulation with PHA, but a
low level of IL-13 in response to A23187 and or PMA. Eosinophils and
neutrophils did not produce IL-13 when cultured with A23187, IL-5, and
anti-Fc epsilon RI alpha.. This is the first demonstration of IL-13
prodn. by basophils. Our data suggest that basophils, in addn. to
secreting mediators, can represent an important source of proallergic
cytokines.

L27 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:30117 CAPLUS
DOCUMENT NUMBER: 126:103048
TITLE: Interleukin-13, in combination with anti-interleukin-12, increases graft prolongation after portal venous immunization with cultured allogeneic bone marrow-derived dendritic cells
AUTHOR(S): Gorczynski, Reginald M.; Cohen, Zane; Fu, Xin Ming; Hua, Zeng; Sun, Yonghang; Chen, Zhiqi
CORPORATE SOURCE: Departments Surgery and Immunology, University Toronto, Toronto, M5G 2C4, Can.
SOURCE: Transplantation (1996), 62(11), 1592-1600
CODEN: TRPLAU; ISSN: 0041-1337
PUBLISHER: Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Portal venous (pv) transfusion before transplant with large nos. (200 tens - 10^6) of irradiated multiple minor histoincompatible spleen cells (BALB/c) augments allogeneic skin graft survival in C3H mice. We have shown in earlier studies that this is correlated with preferential activation for prodn. of type 2 cytokines (interleukin [IL]-4 and IL-10) and decreased prodn. of type 1 cytokines (IL-2 and interferon [IFN]-gamma). We have also shown that recombinant (r) IL-12, in assocn. with anti-IL-12 monoclonal antibody, can reverse in vivo the graft prolongation afforded by pv immunization and the altered cytokine prodn. that follows. Adoptive transfer of inhibition of graft rejection is possible at early times after pv immunization, using plastic adherent cells obtained from the liver of treated mice. We show that within 4 days of pv immunization, dendritic cells (NLDC-145+) isolated from the thymus, mesenteric lymph node (MLN), and spleen of mice receiving MHC incompatible cells/graft (C3H with C57BL/6), can transfer skin graft prolongation to

transplant and manipulation of cytokine levels in vivo may prove an effective regimen in the induction of unresponsiveness in transplant recipients.

=d l26 ibib abs 1-16

L26 ANSWER 1 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:696137 CAPLUS
DOCUMENT NUMBER: 137:231354
TITLE: Method for constructing expression cassette of a chimeric interleukin 13 (IL-13) vaccine and therapeutic uses
INVENTOR(S): Ashman, Claire; Crowe, James Scott; Ellis, Jonathan Henry; Lewis, Alan Peter
PATENT ASSIGNEE(S): Glaxo Group Limited, UK
SOURCE: PCT Int. Appl., 83 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002070711	A1	20020912	WO 2002-GB900	20020301

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: GB 2001-5360 A 20010303

AB The present invention provides a method for constructing expression cassette of a chimeric interleukin 13 (IL-13) vaccine in which the sequence of the predicted antigenic loops has been taken from murine IL-13, and the sequence of the predicted structural (predominantly helical) regions has been taken from human IL-13. The present invention relates to an isolated polypeptide useful for immunization against self antigens. In particular the invention relates to a self-protein that is capable of raising auto-antibodies when administered in vivo. The invention particularly relates to rendering human cytokines immunogenic in humans. The invention further relates to pharmaceutical compns. comprising such compds. and their use in medicine and to methods for their prodn.

REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L26 ANSWER 2 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:343956 BIOSIS
DOCUMENT NUMBER: PREV200203439936
TITLE: A monoclonal antibody to mouse IL-13 inhibits acute asthma response.
AUTHOR(S): Yang, Guoxin (1); Emmell, Eva (1); Sheale, Dave (1); Orr-Weaver, Don (1); Li, Lei (1)
CORPORATE SOURCE: (1) Centocor, Inc., 200 Great Valley Parkway, Malvern, PA, 19355, USA
SOURCE: FASEB Journal, (March 20, 2002) Vol. 16, No. 4, pp. A672.
http://www.faseb.org/meet/
Meeting Info.: Annual Meeting of the Professional Research Scientists on Experimental Biology New Orleans, Louisiana, USA April 20-24, 2002
ISSN: 0892-6638
DOCUMENT TYPE: Conference

therapeutic potential on asthma. To prove the concept, we have developed a
rat anti-mouse IL-13 neutralizing monoclonal antibody and tested its effects on OVA induced acute asthma responses in mice. IL-13 was up-regulated in the lung during OVA induced asthma responses. When administered at the challenge stage, the anti-IL-13 monoclonal antibody significantly inhibited AHR, goblet cell hyperplasia and mucus production.
Furthermore, the antibody treatment also inhibited the production IL-5, IL-6, and eotaxin in the lung. These results clearly demonstrated that IL-13 plays an important role in asthma responses, and suggest that a monoclonal ***antibody*** to ***IL*** - ***13*** would be an
effective therapeutic agent in the treatment of asthma.

L26 ANSWER 3 OF 16 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2002175993 MEDLINE
DOCUMENT NUMBER: 21905224 PubMed ID: 11908556
TITLE: High levels of interleukin 13 in rheumatoid arthritis sera are modulated by tumor necrosis factor antagonist therapy: association with dendritic cell growth activity.
AUTHOR: Tokayer Amiel; Carsons Steven E; Chokshi Binny; Santiago-Schwarz Frances
CORPORATE SOURCE: Winthrop University Hospital, Mineola, New York 11501, USA.
SOURCE: JOURNAL OF RHEUMATOLOGY, (2002 Mar) 29 (3) 454-61.
Journal code: 7501984. ISSN: 0315-162X.
PUB. COUNTRY: Canada
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200209
ENTRY DATE: Entered STN: 20020324
Last Updated on STN: 20020925
Entered Medline: 20020924
AB OBJECTIVE: To investigate the physiology of interleukin 13 (IL-13) in
rheumatoid arthritis (RA) and the effects of tumor necrosis factor (TNF)
antagonists (etanercept) on the distribution of IL-13 in patients with RA.
METHODS: We measured cytokine levels in RA sera (pre post etanercept), RA
synovial fluid (SF), osteoarthritis (OA) SF, and normal human sera by ELISA. Detection of IL-13 was not influenced by rheumatoid factor, as revealed in spike recovery and isotype antibody control studies.
Biologically active IL-13 in RA SF was studied using dendritic cell (DC)
progenitors that develop into mature DC with IL-13 and with neutralizing
antibodies to ***IL*** - ***13*** . The modulation of IL-13
by etanercept was compared to that of IL-6 and monocyte colony stimulating
factor (M-CSF). The effect of etanercept on the ability of RA sera to promote DC growth was studied using DC progenitors. RESULTS: IL-13 was
increased in RA sera versus normal sera, OA SF, and RA SF. Relative to OA
SF and normal sera, RA SF was enriched in IL-13. The IL-13 contained in RA
samples was biologically active, prompting DC growth from progenitors.
Circulating DC growth activity was strongly reduced by anti-TNF therapy.
Whereas decreases in DC growth factors including IL-13 and IL-6 occurred
with etanercept therapy and were associated with clinical improvement, concurrent increases in circulating M-CSF (a non-DC, monocyte-specific
growth factor) were noted. CONCLUSION: The increase of biologically active
IL-13 in RA supports the concept that IL-13 regulates immune cell (including dendritic cell) activity and indicates how the varied anatomical distribution of cytokines may play a role in the RA disease process. The differential regulation of circulating IL-13 and M-CSF levels
by TNF antagonists further implies discrete roles in the TNF-cytokine network in RA.

L26 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:360036 CAPLUS
DOCUMENT NUMBER: 134:365710
TITLE: Modulating IL-13 activity using mutated IL-13 molecules that are antagonists or agonists of IL-13
INVENTOR(S): Puri, Raj K.; Oshima, Yasuo; Joshi, Bharat H
PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA

HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
AU 2001015993 A5 20010606 AU 2001-15993 20001110
PRIORITY APPLN. INFO.: US 1999-165236P P 19991111
WO 2000-US31044 W 20001110
AB The present invention provides antagonists and agonists of IL-13 activity.
The antagonists comprise a mutation in which the glutamic acid at position
13 of the IL-13 mol. is changed to a neutral or a pos. charged mol. The antagonists can be used to reduce or end symptoms in conditions, such as
asthma, allergic rhinitis, atopic dermatitis, and hepatic fibrosis in schistosomiasis, in which IL-13 is an initiator, mediator, or enhancer of the abnormal state. Addnl. the antagonists can be used to slow the growth of cells of cancers for which IL-13 is an autocrine growth factor.
Such cancers include renal cell carcinoma, Kaposi's sarcoma, and Hodgkin's
disease. The agonists comprise mutated IL-13s in which one or more of the
residues at positions 112, 110, 109, 92, 69, or 66 are mutated to a neutrally charged residue, or one with a charge opposite to the charge of
the residue found at that position in native IL-13, provided that the residue at position 13 of the mol. is not neg. charged. The agonists can be used as more potent agents to provoke an effect provided by IL-13. In
particular, the agonists can be used as reagents in the maturation of monocytes into dendritic cells, or to pretreat bone marrow stem cell donors to reduce graft vs. host disease in the recipient of the stem cells. Finally, the invention provides IL-13 receptor binding mols. with affinity for the IL-13 receptor at least about 3 times greater than that exhibited by wild-type IL-13. Also provided are methods and compns. for
specifically delivering an effector mol. to a tumor cell by chimeric mols. comprising the effector mol. and an IL-13 receptor binding mol., and pharmaceutical compns. comprising such chimeric mols.

L26 ANSWER 5 OF 16 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2001255129 MEDLINE
DOCUMENT NUMBER: 21217071 PubMed ID: 11316662
TITLE: Decreased steroid responsiveness at night in nocturnal asthma. Is the macrophage responsible?.
AUTHOR: Kraft M; Hamid Q; Chrousos G P; Martin R J; Leung D Y
CORPORATE SOURCE: Departments of Medicine and Pediatrics, National Jewish
Medical and Research Center, Division of Pulmonary Sciences and Critical Care Medicine, University of Colorado Health Sciences Center, Denver, Colorado, USA.. kraftm@njc.org
CONTRACT NUMBER: AR-41256 (NIAMS)
HL03343 (NHLBI)
HL36577 (NHLBI)
RR-00051 (NCRR)
SOURCE: AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE,
(2001 Apr) 163 (5) 1219-25.
Journal code: 9421642. ISSN: 1073-449X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010611
Last Updated on STN: 20010611
Entered Medline: 20010607
AB As peripheral blood mononuclear cells from patients with nocturnal asthma
(NA) exhibit reduced steroid responsiveness at 4:00 A.M. as compared with
4:00 P.M., we hypothesized that NA is associated with increased nocturnal
airway cell expression of GRbeta, an endogenous inhibitor of steroid action. Ten subjects with NA and seven subjects with nonnocturnal asthma
(NNA) underwent bronchoscopy with bronchoalveolar lavage (BAL) at 4:00
P.M. and 4:00 A.M. BAL [illegible]

but only in the NA group and addition of neutralizing ***antibodies***
to ***IL*** - ***13*** reduced GRbeta expression by BAL macrophages.
We conclude that the airway macrophage may be the airway inflammatory cell
driving the reduction in steroid responsiveness at night in NA, and this function is modulated by IL-13.

L26 ANSWER 6 OF 16 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 2002068968 MEDLINE
DOCUMENT NUMBER: 21653974 PubMed ID: 11795676
TITLE: Childhood asthma as an allergic disease: rationale for the development of future treatment.
AUTHOR: Tang M L; Powell C V
CORPORATE SOURCE: Department of Paediatrics, University of Melbourne, Royal
Children's Hospital, Parkville, Victoria, Australia.
SOURCE: EUROPEAN JOURNAL OF PEDIATRICS, (2001 Dec) 160 (12)
696-704. Ref: 123
Journal code: 7603873. ISSN: 0340-6199.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020321
Entered Medline: 20020320
AB The fundamental abnormality in asthma is inflammation of the airways.
T-helper 2 (Th2) lymphocytes are the key orchestrators of this inflammation, initiating and propagating inflammation through the release
of Th2 cytokines. Interleukins(IL)-4, IL-5 and IL-13. IL-4 and IL-13 promote IgE production by B-cells, mast cell growth and differentiation,
and upregulate adhesion molecule expression on vascular endothelium. IL-4
also promotes differentiation of uncommitted Th0 lymphocytes into Th2 lymphocytes. IL-5 promotes differentiation and recruitment of eosinophils
and activates them to degranulate within tissues, resulting in damage to the respiratory epithelium. Current treatment of childhood asthma relies predominantly on corticosteroids that have nonspecific anti-inflammatory
activity and are associated with potential side-effects. Novel therapies that selectively target the underlying immunopathogenesis hold great promise. Disruption of the Th2 lymphocyte induced allergic inflammatory
response represents a novel approach to selectively inhibiting allergic inflammation at its origin. Possible therapeutic interventions include inhibition of Th2 response (CpG oligonucleotides, vaccination, CTLA4Ig
fusion protein, IL-12, IL-10), inhibition of IgE (the anti-IgE antibody rhuMAb-E25 omalizumab which is undergoing clinical trials), inhibition of
mediator activity (leukotriene modifiers, which are approved for use in childhood asthma), and targeting Th2 cytokines (soluble IL-4 receptors, IL-5 ***antibody*** , ***IL*** - ***13***). Other therapeutic approaches targeting downstream events in the allergic inflammatory cascade are also currently under investigation (chemokine receptors CCR3,
tryptase inhibitors, and inhibitors of cyclic AMP-specific phosphodiesterase 4). CONCLUSION: As we further understand the pathophysiology of asthma, the potential to develop novel treatments increases. This paper addresses current possible new treatments for the future.

L26 ANSWER 7 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:186429 BIOSIS
DOCUMENT NUMBER: PREV200200186429
TITLE: NFkappaB activation in Hodgkin - Reed-Sternberg cells can be
decreased by inhibition of interleukin-13 signalling
AUTHOR(S): Knorre, Alexander (1); Skinnider, Brian F.; Kaiser, Stefan;
Sebald, Walter; Pahl, Heike (1); Mak, Tak W.; Kapp, Ursula
CORPORATE SOURCE: (1) Experimental Anaesthesiology, University Medical
Center, Freiburg Germany
SOURCE: Blood, (November 16, 2001) Vol. 98, No. 11 Part 1, pp.
406a. http://www.bloodjournal.org/. print.
Meeting Info.: 43rd Annual Meeting of the American Society

25
to 100% of HRS cells in 86% of 36 cases of classical HL tested by in situ
hybridisation. Furthermore we were able to demonstrate that proliferation
of the IL-13 secreting cell lines HDLM2 and L1236 can be inhibited by
treatment with IL-13 neutralizing antibodies. These findings suggest that
an autocrine stimulation by IL-13 might be one step in the multistep
transformation process of HL. Another pathway that might play a role for
proliferation and survival of HRS cells is activation of NF-kappaB. As
opposed to normal B cells constitutive presence of NF-kappaB1 re1A could
be demonstrated in the nucleus of HRS-cells. Here we investigate whether
IL-13 signaling and activation of NF-kappaB might be linked to each other
in HL. HL-derived cell lines HDLM2 and L1236 were cultured untreated or in
the presence of different compounds inhibiting IL-13 signaling: IL-13
neutralizing ***antibodies*** (alpha- ***IL*** - ***13***),
specific antibodies blocking the IL-13/IL-4 receptor (alpha-IL13/IL4R) and
an IL-4 mutant molecule (IL4RY). After 48h of treatment cells were
harvested and investigated for nuclear NF-kappaB1 re1A by gel-shift and
super-shift experiments. At the same time, treated cells were also tested
for cell proliferation by measurement of (3H)-thymidine uptake. In both
cell lines treatment with alpha-IL-13, alpha-IL13/IL4R and IL4RY inhibited
proliferation. In HDLM2 cells neutralization of IL-13, as well as blocking
of the IL-13/IL-4R leads to a significant loss of nuclear
NF-kappaB1 re1A.
In L1236 NF-kappaB activation was not altered by IL-13 neutralization.
This study indicates that NF-kappaB1 re1A activation may be linked to
IL-13 signalling mediated by the IL-13/IL-4R in HL-derived cells.
Proliferation of the cell line L1236 can be inhibited by IL-13
neutralization without inactivation of NF-kappaB1 re1A, which suggests
that the proliferative effect of IL-13 on HL-cells might not depend on
NF-kappaB activation.

L26 ANSWER 8 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:129980 BIOSIS
DOCUMENT NUMBER: PREV200200129980
TITLE: Interleukin 13 (IL-13) levels in serum from patients with Hodgkin disease (HD) and healthy volunteers.
AUTHOR(S): Fiumara, Paolo (1); Cabanillas, Fernando (1); Younes, Anas (1)
CORPORATE SOURCE: (1) Lymphoma Myeloma, M.D. Anderson Cancer Center, Houston, TX USA
SOURCE: Blood, (November 16, 2001) Vol. 98, No. 11 Part 1, pp. 129a. http://www.bloodjournal.org/. print.
Meeting Info.: 43rd Annual Meeting of the American Society of Hematology. Part 1 Orlando, Florida, USA December 07-11, 2001
ISSN: 0006-4971.
DOCUMENT TYPE: Conference
LANGUAGE: English
AB Interleukin 13 (IL-13) has recently been found to be highly expressed in cultured Hodgkin disease (HD)-derived cell lines and primary Hodgkin and Reed-Sternberg (HRS) cells. Furthermore, IL-13 has been detected in the supernatants of HD-derived cell lines by enzyme linked immunosorbent assay (ELISA), and neutralizing ***antibody*** to ***IL*** ***13*** results in inhibition of HRS cell proliferation in vitro. Because of the potential therapeutic implication of these observations, we examined IL-13 levels in serum from patients with newly diagnosed and relapsed HD and healthy volunteers. Supernatants from 3 HD-derived cell lines (HD-LM2, L-428, and KMH-2) known to produce IL-13 were used as positive controls. The sensitivity of the ELISA assay is less than 12 pg/ml. As previously reported, all 3 HD cell lines produced IL-13 (range 85-500 pg/ml). In contrast, IL-13 was below the detectable level in sera from 40 healthy disease-free subjects. Of the 13 newly diagnosed patients who had elevated serum IL-13 levels, only one patient experienced disease progression on 4

for
the first time that IL-13 levels can be elevated in the serum of patients with HD. Although the number of patients with elevated IL-13 levels is small, this does not rule out the possibility of higher concentrations at the disease site. Our data may serve as the basis for new treatment strategies to explore the potential clinical relevance of IL-13 in patients with HD.

L26 ANSWER 9 OF 16 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2001-080753 [09] WPIDS
DOC. NO. CPI: C2001-023298
TITLE: Treating tissue fibrosis and or inhibiting formation of tissue fibrosis in a mammalian subject, involves administering a pharmaceutical composition comprising IL-13 antagonist.
DERWENT CLASS: B04
INVENTOR(S): COLLINS, M; DONALDSON, D; FITZ, L; NEBEN, T; WHITTERS, M
J; WILLS-KARP, M; WOOD, C
PATENT ASSIGNEE(S): (GEMY) GENETICS INST INC; (UYJO) UNIV JOHNS HOPKINS
COUNTRY COUNT: 83
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000078336	A1	20001228	(200109)*	EN	72

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZW
AU 2000057561 A 20010109 (200122)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000078336	A1	WO 2000-US17103	20000621
AU 2000057561	A	AU 2000-57561	20000621

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000057561	A Based on	WO 200078336

PRIORITY APPLN. INFO: US 1999-334512 19990621
AN 2001-080753 [09] WPIDS
AB WO 200078336 A UPAB: 20010213
NOVELTY - Treating tissue fibrosis and or inhibiting formation of tissue
fibrosis in a mammalian subject, comprising administering a pharmaceutical composition (C1) comprising a protein (I), or a composition (C2) comprising a molecule (II) which is interleukin (IL)-13 or IL-4 antagonist, is new.
DETAILED DESCRIPTION - Treating tissue fibrosis and or inhibiting formation of tissue fibrosis in a mammalian subject, comprising administering a pharmaceutical composition (C1) comprising a protein (I), or a composition (C2) comprising a molecule (II) which is interleukin (IL)-13 or IL-4 antagonist, is new. (I) comprises a 383 residue amino acid sequence (S1), fully defined in the specification, residues 22-334 or 355-383 of S1, a 380 residue amino acid sequence (S2), fully defined in the specification, amino acids 26-341 or 363-380 of S2, or fragments of S1 or S2 having a biological activity of IL-13 receptor binding chain.
ACTIVITY - Cytostatic.
MECHANISM OF ACTION - Inhibitor of tissue fibrosis formation (claimed).
C57BL/6 WT and IL-4-deficient mice were infected percutaneously with 25 Schistosoma mansoni cercariae. Separate groups of animals were treated with either sIL-13R alpha 2-Fc or with control-Fc. The treatments began on week 5, at the start of egg laying, and all animals were sacrificed 8 week post-infection and examined for several parasitologic and immunologic parameters. All four groups of mice harbored similar worm burdens, and

L26 ANSWER 10 OF 16 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2001-024676 [03] WPIDS
DOC. NO. CPI: C2001-007458
TITLE: Treating or inhibiting tissue fibrosis resulting from infection with schistosoma and wound healing involves administering interleukin-13 or interleukin-4 antagonist.
DERWENT CLASS: B04
INVENTOR(S): CHIARAMONTE, M G; COLLINS, M; DONALDSON, D; FITZ, L; NEBEN, T; WHITTERS, M J; WOOD, C; WYNN, T A
PATENT ASSIGNEE(S): (GEMY) GENETICS INST INC; (GEMY) GENETICS INST LLC
COUNTRY COUNT: 83
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000064944	A1	20001102	(200103)*	EN	82

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZW
AU 2000046805 A 20001110 (200109)
EP 1173484 A1 20020123 (200214) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
CN 1348465 A 20020508 (200253)
HU 2002000862 A2 20020729 (200258)
KR 2002026426 A 20020410 (200267)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000064944	A1	WO 2000-US11612	20000428
AU 2000046805	A	AU 2000-46805	20000428
EP 1173484	A1	EP 2000-928591	20000428
		WO 2000-US11612	20000428
CN 1348465	A	CN 2000-806772	20000428
HU 2002000862	A2	WO 2000-US11612	20000428
		HU 2002-862	20000428
KR 2002026426	A	KR 2001-713820	20011029

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000046805	A Based on	WO 200064944
EP 1173484	A1 Based on	WO 200064944
HU 2002000862	A2 Based on	WO 200064944

PRIORITY APPLN. INFO: US 1999-301808 19990428
AN 2001-024676 [03] WPIDS
AB WO 200064944 A UPAB: 20010116
NOVELTY - Treating or inhibiting formation of tissue fibrosis in a mammalian subject, comprises administering a composition comprising an interleukin(IL)-13 antagonist or an IL-4 antagonist.
ACTIVITY - Vulnerary. The effect of IL-13 inhibitor antagonist such as soluble IL-13R alpha 2-Fc in preventing fibrosis associated with chronic infectious diseases was studied. C57BL/6 WT and IL-4 deficient mice were infected percutaneously with Schistosoma mansoni cercariae. Separate groups of animals were treated with either sIL-13R alpha 2-Fc or with control Fc. All animals were sacrificed 8 week postinfection and examined for several parasitologic and immunologic parameters. WT mice

animals, and more than a 75 % reduction when compared with control-Fc-treated IL-4-deficient mice.
USE - (I) is useful for treating tissue fibrosis resulting from infection with Schistosoma or from healing of a wound which is a surgical incision, and or inhibiting formation of tissue fibrosis which affects tissues such as liver, skin epidermis, skin endodermis, muscle, tendon, cartilage, cardiac tissue, pancreas, lung, uterine tissue, neural tissue, testes, ovary, adrenal gland, artery, vein, colon, small intestine, biliary tract and gut (claimed).
Dwg.0/7

IL-4-deficiency
resulted in a less significant reduction. The overall results showed that treatment with -IL-13R alpha 2-Fc significantly reduced hepatic fibrosis in S.mansoni-infected mice.

MECHANISM OF ACTION - IL-13 or IL-4 inhibitor antagonist.

USE - The method is useful for treating or inhibiting the formation of tissue fibrosis resulting from infection with schistosoma or from healing of a surgical incision wound. Fibrosis affects skin epidermis, skin endodermis, muscle, tendon, cartilage, tissues of cardiac, pancreatic, lung, uterine, neural, testis, ovary, adrenal gland, artery, vein, colon, small intestine, biliary tract or gut tissue or more preferably liver tissue (claimed).

Dwg.0/7

L26 ANSWER 11 OF 16 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2000-431587 [37] WPIDS
DOC. NO. CPI: C2000-131254
TITLE: New polynucleotide encoding an interleukin-13 (IL-13) binding chain of an IL-13 receptor for treating IgE-mediated conditions, such as atopy, asthma, Grave's disease and inflammatory conditions of the lung.
DERWENT CLASS: B04 D16
INVENTOR(S): COLLINS, M; DONALDSON, D; FITZ, L; NEBEN, T; WHITTERS, M J; WILLS-KARP, M; WOOD, C
PATENT ASSIGNEE(S): (GEMY) GENETICS INST INC; (UYJO) UNIV JOHNS HOPKINS
COUNTRY COUNT: 83
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000036103	A1	20000622	(200037)*	EN	60

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZW

PATENT NO	KIND	DATE	WEEK	LA	PG
AU 2000021775	A	20000703	(200046)		
EP 1141286	A1	20011010	(200167)	EN	

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI

PATENT NO	KIND	DATE	WEEK	LA	PG
BR 9916209	A	20011226	(200206)		
CN 1352686	A	20020605	(200261)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000036103	A1	WO 1999-US29493	19991213
AU 2000021775	A	AU 2000-21775	19991213
EP 1141286	A1	EP 1999-966166	19991213
		WO 1999-US29493	19991213
BR 9916209	A	BR 1999-16209	19991213
		WO 1999-US29493	19991213
CN 1352686	A	CN 1999-815591	19991213

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000021775	A	Based on	WO 200036103
EP 1141286	A1	Based on	WO 200036103
BR 9916209	A	Based on	WO 200036103

PRIORITY APPLN. INFO: US 1998-211335 19981214
AN 2000-431587 [37] WPIDS
AB WO 200036103 A UPAB: 20000807

NOVELTY - A polynucleotide comprising a nucleotide sequence that encodes an interleukin-13 binding chain (IL-13bc) or fragment, of IL-13 receptor, is new.

DETAILED DESCRIPTION - The polynucleotide comprises a nucleotide sequence that is:

(a) nucleotides 256 to 1404 of a 1525 murine nucleotide sequence, given in the specification;

(b) nucleotides 103 to 1242 of a 1369 human nucleotide sequence, given in the specification;

(c) a variant of (a) or (b) as a result of degeneracy of the genetic code;

(VI) amino acids 363 to 380 of (IV); or

(VII) fragments of (I) to (VI) having IL-13 receptor binding chain activity;

(4) a protein produced by (2);

(5) a composition comprising an antibody that reacts with (3);

(6) identifying an inhibitor of IL-13 binding to the IL-13 receptor (IL-13R) comprising:

(i) combining (2) with IL-13 or a fragment to form a first binding mixture;

(ii) measuring binding between the protein and IL-13 or fragment;

(iii) combining a compound with the protein and IL-13 or fragment to form a second binding mixture;

(iv) measuring the amount of binding; and

(v) comparing the binding in the first binding mixture with the binding in the second binding mixture, where the compound inhibits IL-13 binding to IL-13R when there is a decrease in the binding of the second binding mixture;

(7) an inhibitor identified by (6);

(8) inhibiting binding of IL-13 to IL-13R in a mammal comprising administering (7), (3) or (5);

(9) a polynucleotide comprising a nucleotide sequence that encodes a peptide or protein with an amino acid sequence of (3);

(10) treating an IL-13-related condition in a mammal by administering (3) or an IL-13 antagonist;

(11) potentiating IL-13 activity comprising combining a protein with IL-13 activity with (3) and contacting the combination with a cell expressing a chain of IL-12R other then IL-13bc; and

(12) inhibiting interaction of IL-13 with IL-13bc in a mammal by administering IL-13 antagonist.

ACTIVITY - Antiallergic; antiinflammatory; antiasthmatic; dermatological; immunosuppressive; antithyroid; cytostatic.

Male A/J mice were immunized intraperitoneally and challenged intratracheally with soluble ovalbumin. The allergic phenotype was assessed 4 days after the antigen challenge. Blockade of IL-13 was performed 24 hours before the allergen challenge by systemic administration of soluble IL-13bc-IgGFc fusion protein which binds to and neutralizes IL-13. Challenge of allergen-immunized mice resulted in significant increases in airway responsiveness to acetylcholine. Blockade of IL-13 resulted in complete reversal of the established allergen-induced airway hyper responsiveness, showing that asthma may be treated.

MECHANISM OF ACTION - IL-13 inhibitor.

USE - For identifying and producing an IL-13bc protein that can inhibit the binding of IL-13 to an IL-13 receptor and treat an IL-13-related condition such as an IgE-mediated condition. Atopy, allergic conditions, asthma, immune complex diseases, lupus, nephritis, thyroiditis, Grave's disease or inflammatory conditions of the lung can be treated. For potentiating IL-13 activity (all claimed). Cancer may be treated. Macrophage activation is enhanced allowing use in vaccination and treatment of mycobacterial, intracellular organisms, or parasitic infections.

Dwg.0/4

L26 ANSWER 12 OF 16 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1999307279 MEDLINE
DOCUMENT NUMBER: 99307279 PubMed ID: 10377189
TITLE: Interleukin 13 is secreted by and stimulates the growth of Hodgkin and Reed-Sternberg cells.
AUTHOR: Kapp U; Yeh W C; Patterson B; Elia A J; Kagi D; Ho A; Hessel A; Tipsword M; Williams A; Mirtsos C; Itie A; Moyle M; Mak T W
CORPORATE SOURCE: Amgen Institute, Ontario Cancer Institute, the Department of Medical Biophysics, and the Department of Immunology, University of Toronto, Toronto, Ontario M5G 2C1, Canada.
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1999 Jun 21) 189 (12) 1939-46.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990806
Last Updated on STN: 19990806
Entered Medline: 19990726

AB Gene expression patterns can provide vital clues to the pathogenesis of neoplastic diseases. We investigated the expression of 950 genes in [illegible]

showed that elevated levels of IL-13 were specifically expressed by Hodgkin Reed-Sternberg (H/RS) tumor cells. Treatment of a HD-derived cell line with a neutralizing ***antibody*** to ***IL*** - ***13*** resulted in a dose-dependent inhibition of H/RS cell proliferation. These data suggest that H/RS cells produce IL-13 and that IL-13 plays an important role in the stimulation of H/RS cell growth, possibly by an autocrine mechanism. Modulation of the IL-13 signaling pathway may be a logical objective for future therapeutic strategies.

L26 ANSWER 13 OF 16 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 1999333604 MEDLINE
DOCUMENT NUMBER: 99333604 PubMed ID: 10404009
TITLE: A novel T cell cytokine stimulates interleukin-6 in human osteoblastic cells.
AUTHOR: Rifas L; Avioli L V
CORPORATE SOURCE: Department of Internal Medicine, Division of Bone and Mineral Diseases, Washington University School of Medicine at Barnes-Jewish Hospital, St. Louis, Missouri 63110, USA.
SOURCE: JOURNAL OF BONE AND MINERAL RESEARCH, (1999 Jul) 14 (7) 1096-103.
Journal code: 8610640. ISSN: 0884-0431.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199909
ENTRY DATE: Entered STN: 19991012
Last Updated on STN: 19991012
Entered Medline: 19990928

AB Rheumatoid arthritis (RA) is an autoimmune disease characterized by a heavy lymphocytic infiltration into the synovial cavity, resulting in the secretion of a variety of cytokines which ultimately leads to destruction of joint tissue. Among the infiltrating cells are activated T cells which produce specific cytokines capable of osteoclast progenitor cell expansion, fusion, and activation. Cultures of activated human T cells and human osteoblasts (hOBs) were used to study the possibility that lymphokines may act on osteoblasts to produce the osteoclastogenic factor interleukin-6 (IL-6). Purified T cells were activated with a combination of anti-CD3 and anti-CD28 antibodies, cocultured with hOBs in direct physical contact or separated by a transwell system, and conditioned media (CM) were assayed for IL-6 production. After a 72 h incubation period, activated T cell-hOB interaction resulted in a 100-fold increase of IL-6 production over basal levels. The immunosuppressant cyclosporine A (CsA) inhibited T cell tumor necrosis factor alpha and IL-6 production but did not inhibit the T cell induction of IL-6 from hOB. Assay of activated T-cell CM on hOB revealed that a soluble factor, not cell-cell contact, was the major inducer of IL-6. The induction of IL-6 mRNA by both activated T cell CM and CsA-treated activated T cell CM was confirmed by Northern blot analysis. Neutralizing ***antibodies*** to ***IL*** - ***13*** and IL-17 did not affect IL-6 production. These findings suggest that activated T cells produce a novel, potent, IL-6 inducing factor that may be responsible for the bone loss observed in RA patients.

L26 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:433168 CAPLUS
DOCUMENT NUMBER: 121:33168
TITLE: Human interleukin-13 and the gene encoding it
INVENTOR(S): Aversa, Gregorio; Banchereau, Jacques; Briere, Francine; Cocks, Benjamin G.; Coffman, Robert L.; Culpepper, Janice; Dang, Warren; De Vries, Jan; De Waal, Malefyt Rene; et al.
PATENT ASSIGNEE(S): Schering Corp., USA
SOURCE: PCT Int. Appl., 136 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO	KIND	DATE	APPLICATION NO.	DATE
WO 9404680	A1	19940303	WO 1993-US7645	19930818

W: AU, BB, BG, BR, BY, CA, CZ, FI, HU, JP, KR, KZ, LK, MG, MN, MW, NO, NZ, PL, RO, RU, SD, SK, UA, VN
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL

state

(3) an isolated IL-13bc protein comprising a sequence of [illegible] amino acids given in the specification

revealed the expression of IL-13 in a third HD derived cell line, HDLM2. Control

US 1993-[illegible]	1993[illegible]
US 1993-12843	19930201
WO 1993-US7645	19930818

the
cDNA and protein in diagnostics and therapeutics are described. A cDNA
for the protein was cloned from a T-cell cDNA libraries by repeated
screening with a cDNA for mouse P600 protein to obtain overlapping clones
from which a full-length cDNA was constructed. The protein was manufd. as
a fusion protein with glutathione-S-transferase and purified from
inclusion bodies by solubilization, refolding, and cleavage with thrombin.
Human IL-13 stimulated B-cell DNA synthesis through the antigen receptor
and acted as a growth factor for B cells stimulated through the CD40
antigen. IL-13 also stimulated IgE secretion in anti-CD40 activated B
cells. The biol. effects of IL-13 are independent of those of IL-4 and
the target B-cell sub-population is more restricted than that for IL-4.

L26 ANSWER 15 OF 16 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 94265839 MEDLINE
DOCUMENT NUMBER: 94265839 PubMed ID: 7911424
TITLE: Interleukin-13 alters the activation state of murine macrophages in vitro: comparison with interleukin-4 and interferon-gamma.
AUTHOR: Doyle A G; Herbein G; Montaner L J; Minty A J; Caput D; Ferrara P; Gordon S
CORPORATE SOURCE: Sir William Dunn School of Pathology, University of Oxford.
SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (1994 Jun) 24 (6) 1441-5.
Journal code: 1273201. ISSN: 0014-2980.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199407
ENTRY DATE: Entered STN: 19940721
Last Updated on STN: 19990129
Entered Medline: 19940708
AB Interleukin (IL)-13 is a newly described cytokine expressed by activated
lymphocyte. We examined the effects of the murine recombinant cytokine on
the phenotype and activation status of elicited peritoneal macrophages (M
phi) concentrating on activities which are known to be modulated by
interferon-gamma and IL-4. IL-13 markedly suppressed nitric oxide release
and to a lesser extent secretion of the pro-inflammatory cytokine tumor
necrosis factor-alpha. However, anti-microbial capacity was not completely
jeopardized as the respiratory burst was unaffected, and indeed the
enhanced expression of M phi mannose receptor and major histocompatibility
class II, and regulation of sialoadhesin, the M phi sialic acid-specific
receptor involved in hemopoietic and lymphoid interactions, suggest that
these cells are not simply deactivated, but primed for an active role in
immune and inflammatory responses. These activities closely mimic those of
IL-4 but mediation of the effects by IL-4 was discounted by the use of a
neutralizing monoclonal ***antibody***. Thus, ***IL*** - ***13***
, like IL-4, is a cytokine which has complex effects on M phi behavior,
inducing activities characteristic of both activation and deactivation.

L26 ANSWER 16 OF 16 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 95137668 MEDLINE
DOCUMENT NUMBER: 95137668 PubMed ID: 7530690
TITLE: IL-13 has only a subset of IL-4-like activities on B chronic lymphocytic leukaemia cells.
AUTHOR: Buckley A G; Briere F; Zurawski G; Bridon J M; Banchereau J
CORPORATE SOURCE: Schering-Plough Laboratory for Immunological Research, Dardilly, France.
SOURCE: IMMUNOLOGY, (1994 Nov) 83 (3) 397-403.
Journal code: 0374636. ISSN: 0019-2805.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199503
ENTRY DATE: Entered STN: 19950214
Last Updated on STN: 19960129
[illegible]

DNA synthesis. Moreover, IL-13, like IL-4, was able to increase CD23 expression on anti-CD40-activated leukaemic B cells. The CD23 up-regulation and the DNA synthesis induced by IL-13 on anti-CD40-activated B-CLL cells, were significantly reduced when B-CLL
cells were cultured with anti-IL-4 receptor monoclonal antibody, suggesting a common pathway for IL-13 and IL-4 signalling. However, after
cross-linking of surface IgM, IL-4 strongly inhibited the IL-2-induced DNA
synthesis of B-CLL cells, whereas IL-13 did not inhibit IL-2-driven proliferation of anti-IgM-activated B-CLL cells. Furthermore, while IL-4
strongly up-regulated the expression of CD23 on anti-IgM-activated leukaemic B cells, IL-13 only marginally increased it. Finally, IL-13, in contrast to IL-4, did not prevent the entry of B-CLL cells into apoptosis.
Thus IL-13 and IL-4 display comparable effects on anti-CD40-activated B-CLL cells, which are blocked by anti-IL-4 receptor (IL-4R) monoclonal
antibodies. However, ***IL*** - ***13*** -dependent effects
are absent or inefficient in non-activated or anti-IgM-activated B-CLL cells. This suggests that such cells may lack functional IL-13 receptors, though IL-13R and IL-4R on B-CLL cells share a common component.

=d l25 ibib abs 1-7

L25 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:696137 CAPLUS
DOCUMENT NUMBER: 137:231354
TITLE: Method for constructing expression cassette of a chimeric interleukin 13 (IL-13) vaccine and therapeutic uses
INVENTOR(S): Ashman, Claire; Crowe, James Scott; Ellis, Jonathan Henry; Lewis, Alan Peter
PATENT ASSIGNEE(S): Glaxo Group Limited, UK
SOURCE: PCT Int. Appl., 83 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002070711	A1	20020912	WO 2002-GB900	20020301

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: GB 2001-5360 A 20010303
AB The present invention provides a method for constructing expression cassette of a chimeric interleukin 13 (IL-13) vaccine in which the sequence of the predicted antigenic loops has been taken from murine IL-13, and the sequence of the predicted structural predominantly helical regions has been taken from human IL-13. The present invention
relates to an isolated polypeptide useful for immunization against self antigens. In particular the invention relates to a self-protein that can sparingly transiently autoantibodies when administered in vivo. The invention particularly relates to rendering human cytokines immunogenic in
human. The invention further relates to pharmaceutical compns. comprising such compds. and their use in medicine and to methods for their
prodn.
REFERENCE COUNT: 15 THERE ARE 15 CITED
REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE
FORMAT

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200209
ENTRY DATE: Entered STN: 20020324
Last Updated on STN: 20020925
Entered Medline: 20020924
AB OBJECTIVE: To investigate the physiology of interleukin 13 (IL-13) in
rheumatoid arthritis (RA) and the effects of tumor necrosis factor (TNF)
antagonists (etanercept) on the distribution of IL-13 in patients with RA.
METHODS: We measured cytokine levels in RA sera (pre-post etanercept), RA
synovial fluid (SF), osteoarthritis (OA) SF, and normal human sera by ELISA. Detection of IL-13 was not influenced by rheumatoid factor, as revealed in spike recovery and isotype antibody control studies.
Biologically active IL-13 in RA SF was studied using dendritic cell (DC)
progenitors that develop into mature DC with IL-13 and with neutralizing
antibodies to ***IL*** - ***13***. The modulation of IL-13
by etanercept was compared to that of IL-6 and monocyte colony stimulating
factor (M-CSF). The effect of etanercept on the ability of RA sera to promote DC growth was studied using DC progenitors. RESULTS: IL-13 was
increased in RA sera versus normal sera, OA SF, and RA SF. Relative to OA
SF and normal sera, RA SF was enriched in IL-13. The IL-13 contained in RA
samples was biologically active, prompting DC growth from progenitors.
Circulating DC growth activity was strongly reduced by anti-TNF therapy.
Whereas decreases in DC growth factors including IL-13 and IL-6 occurred
with etanercept therapy and were associated with clinical improvement, concurrent increases in circulating M-CSF (a non-DC, monocyte-specific
growth factor) were noted. CONCLUSION: The increase of biologically active
IL-13 in RA supports the concept that IL-13 regulates immune cell (including dendritic cell) activity and indicates how the varied anatomical distribution of cytokines may play a role in the RA disease process. The differential regulation of circulating IL-13 and M-CSF levels
by TNF antagonists further implies discrete roles in the TNF-cytokine network in RA.

L25 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:360036 CAPLUS
DOCUMENT NUMBER: 134:365716
TITLE: Modulating IL-13 activity using mutated IL-13 molecules that are antagonists or agonists of IL-13
INVENTOR(S): Puri, Raj K.; Oshima, Yasuo; Joshi, Bharat H.
PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA
SOURCE: PCT Int. Appl., 129 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001034645	A2	20010517	WO 2000-US31044	20001110
WO 2001034645	A3	20020307		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
AU 2001015993 A5 20010606 AU 2001-15993 20001110
PRIORITY APPLN. INFO.: US 1999-165236P P 19991110
WO 2000-US31044 W 20001110

[illegible]

[illegible]

[illegible]

the
residues at positions 112, 110, 109, 92, 69, or 66 are mutated to a neutrally charged residue, or one with a charge opposite to the charge of
the residue found at that position in native IL-13, provided that the residue at position 13 of the mol. is not neg. charged. The agonists can be used as more potent agents to provoke an effect provided by IL-13. In
particular, the agonists can be used as reagents in the maturation of monocytes into dendritic cells, or to pretreat bone marrow stem cell donors to reduce graft vs. host disease in the recipient of the stem cells. Finally, the invention provides IL-13 receptor binding mols. with affinity for the IL-13 receptor at least about 3 times greater than that exhibited by wild-type IL-13. Also provided are methods and compns. for
specifically delivering an effector mol. to a tumor cell by chimeric mols. comprising the effector mol. and an IL-13 receptor binding mol., and pharmaceutical compns. comprising such chimeric mols.

L25 ANSWER 4 OF 7 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2001255129 MEDLINE
DOCUMENT NUMBER: 21217071 PubMed ID: 11316662
TITLE: Decreased steroid responsiveness at night in nocturnal asthma: Is the macrophage responsible?.
AUTHOR: Kraft M; Hamid Q; Chrousos G P; Martin R J; Leung D Y
CORPORATE SOURCE: Departments of Medicine and Pediatrics, National Jewish
Medical and Research Center, Division of Pulmonary Sciences and Critical Care Medicine, University of Colorado Health Sciences Center, Denver, Colorado, USA. kraftm@njc.org
CONTRACT NUMBER: AR-41256 (NIAMS)
HL03343 (NHLBI)
HL36577 (NHLBI)
RR-00051 (NCRR)
SOURCE: AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE, (2001 Apr) 163 (5) 1219-25.
Journal code: 9421642. ISSN: 1073-449X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010611
Last Updated on STN: 20010611
Entered Medline: 20010607
AB As peripheral blood mononuclear cells from patients with nocturnal asthma (NA) exhibit reduced steroid responsiveness at 4:00 A.M. as compared with 4:00 P.M., we hypothesized that NA is associated with increased nocturnal airway cell expression of GRbeta, an endogenous inhibitor of steroid action. Ten subjects with NA and seven subjects with nonnocturnal asthma (NNA) underwent bronchoscopy with bronchoalveolar lavage (BAL) at 4:00 P.M. and 4:00 A.M. BAL lymphocytes and macrophages were incubated with dexamethasone (DEX) at 10(-5) to 10(-8) M. DEX suppressed proliferation of BAL lymphocytes similarly at 4:00 P.M. and 4:00 A.M. in both groups. However, BAL macrophages from NA exhibited less suppression of IL-8 and TNF-alpha production by DEX at 4:00 A.M. as compared with 4:00 P.M. (p < 0.0001), whereas in the NNA group DEX suppressed IL-8 and TNF-alpha production equally at both time points. GRbeta expression was increased at night only in NA, primarily due to significantly increased expression by BAL macrophages (p = 0.0063). IL-13 mRNA expression was increased at night, but only in the NA group, and addition of neutralizing ***antibodies*** to ***IL*** - ***13*** reduced GRbeta expression by BAL macrophages. We conclude that the airway macrophage may be the airway inflammatory cell driving the reduction in steroid responsiveness at night in NA, and this function is modulated by IL-13.

L25 ANSWER 5 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
ACCESSION NUMBER: 2002186429 BIOSIS
DOCUMENT NUMBER: PREV200218642 9
TITLE: NFkappaB activation in Hodgkin- Reed-Sternberg cells [illegible]
SOURCE: Blood, (November 16, 2001) Vol. 98, No. 11, Part pp.
[illegible] http://www.bloodjournal.org/ print
Meeting Info.: 43rd Annual Meeting of the American Society [illegible]

DOCUMENT TYPE: Conference
LANGUAGE: English
AB The unique cellular background of reactive cells surrounding the rare population of Hodgkin- Reed-Sternberg (HRS) cells in Hodgkin specimen and
the systemic clinical symptoms of Hodgkin lymphoma (HL) suggest that cytokines play a role in the pathogenesis of the disease. We have demonstrated previously that interleukin (IL)-13 is strongly expressed and
secreted by some Hodgkin-derived cell lines and also expressed by HRS cells in primary tissue. Specific expression of IL-13 could be found in 25
to 100% of HRS cells in 86% of 36 cases of classical HL tested by in situ
hybridisation. Furthermore we were able to demonstrate that proliferation
of the IL-13 secreting cell lines HDLM2 and L1236 can be inhibited by treatment with IL-13 neutralizing antibodies. These findings suggest that
an autocrine stimulation by IL-13 might be one step in the multistep transformation process of HL. Another pathway that might play a role for
proliferation and survival of HRS cells is activation of NF-kappaB. As opposed to normal B cells constitutive presence of NF-kappaB1 relA could
be demonstrated in the nucleus of HRS-cells. Here we investigate whether
IL-13 signalling and activation of NF-kappaB might be linked to each other
in HL. HL-derived cell lines HDLM2 and L1236 were cultured untreated or in
the presence of different compounds inhibiting IL-13 signalling: IL-13 neutralizing ***antibodies*** (alpha- ***IL*** - ***13***), specific antibodies blocking the IL-13 IL-4 receptor (alpha-IL13 IL4R) and
an IL-4 mutant molecule (IL4KY). After 48h of treatment cells were harvested and investigated for nuclear NF-kappaB1 relA by gel-shift and
super-shift experiments. At the same time, treated cells were also tested for cell proliferation by measurement of (3H)-thymidine uptake. In both cell lines treatment with alpha-IL-13, alpha-IL13 IL4R and IL4KY inhibited
proliferation. In HDLM2 cells neutralization of IL-13, as well as blockade
of the IL-13 IL-4R leads to a significant loss of nuclear NF-kappaB1 relA.
In L1236 NF-kappaB activation was not altered by IL-13 neutralization. This study indicates that NF-kappaB1 relA activation may be linked to IL-13 signalling mediated by the IL-13 IL-4R in HL-derived cells. Proliferation of the cell line L1236 can be inhibited by IL-13 neutralization without inactivation of NF-kappaB1 relA, which suggests
that the proliferative effect of IL-13 on HL-cells might not depend on NF-kappaB activation.

L25 ANSWER 6 OF 7 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1999333604 MEDLINE
DOCUMENT NUMBER: 99333604 PubMed ID: 10404089
TITLE: A novel T cell cytokine stimulates interleukin-6 in human osteoblastic cells.
AUTHOR: Rifas L; Avioli L V
CORPORATE SOURCE: Department of Internal Medicine, Division of Bone and
Mineral Diseases, Washington University School of Medicine at Barnes-Jewish Hospital, St. Louis, Missouri 63110, USA.
SOURCE: JOURNAL OF BONE AND MINERAL RESEARCH, (1999 Jul) 14 (7) 1096-103.
Journal code: 8610640. ISSN: 0884-0431.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199909
ENTRY DATE: Entered STN: 19991012
Last Updated on STN: 19991012
Entered Medline: 19990928
AB The inflamed arthritic (IA) rat is an autoimmune disease characterized by a
heavy lymphocytic infiltration into the synovial cavity, resulting in the secretion of a variety of cytokines which ultimately leads to destruction of joint tissue. Among the infiltrating cells are activated T cells which produce specific cytokines capable of osteoclast progenitor cell expansion, fusion, and activation. Cultures of activated human T cells and
human osteoblasts (hOBs) were used to study the possibility that lymphokines may act on osteoblasts to produce the osteoclastogenic factor
[illegible] T cells were activated with a combination of
[illegible] factor-alpha and IL-6 production but did not [illegible] the T cell induction of IL-6 from hOB. Assays of activated T cell-CM on hOB revealed that a soluble factor, not cell-cell contact, was the major inducer of IL-6. The induction of IL-6 mRNA by both
[illegible]

13 and IL-17 did not affect IL-6 production. These findings suggest that activated T cells produce a novel, potent, IL-6 inducing factor that may be responsible for the bone loss observed in RA patients.

L25 ANSWER 7 OF 7 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 95137668 MEDLINE
DOCUMENT NUMBER: 95137668 PubMed ID: 7530690
TITLE: IL-13 has only a subset of IL-4-like activities on B chronic lymphocytic leukaemia cells.
AUTHOR: Fluckiger A C; Briere F; Zurawski G; Bridon J M; Banchereau J
CORPORATE SOURCE: Schering-Plough, Laboratory for Immunological Research, Dardilly, France.
SOURCE: IMMUNOLOGY, (1994 Nov) 83 (3) 397-403.
Journal code: 0374672. ISSN: 0019-2805.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199503
ENTRY DATE: Entered STN: 19950314
Last Updated on STN: 19960129
Entered Medline: 19950302
AB The recently described interleukin-13 (IL-13) has been shown to share many
of the effects of IL-4 on normal B cells, including growth-promoting activity and induction of CD23. In this study, we compared the effects of
IL-13 and IL-4 on B chronic lymphocytic leukaemias (B-CLL) cells. After
anti-CD40 activation, both IL-13 and IL-4 promoted the DNA synthesis of
B-CLL cells and increased the recovery of viable cells. The time kinetic of the proliferative response of B-CLL cells to IL-13 or IL-4 were superimposable and showed the long-lasting effect of both cytokines. As on
normal B cells, both IL-4 and IL-13 synergized with IL-10 to enhance B-CLL
DNA synthesis. Moreover, IL-13, like IL-4, was able to increase CD23 expression on anti-CD40-activated leukaemic B cells. The CD23 up-regulation and the DNA synthesis induced by IL-13 on
anti-CD40-activated B-CLL cells, were significantly reduced when B-CLL
cells were cultured with anti-IL-4 receptor monoclonal antibody, suggesting a common pathway for IL-13 and IL-4 signalling. However, after
cross-linking of surface IgM, IL-4 strongly inhibited the IL-2-induced DNA
synthesis of B-CLL cells whereas IL-13 did not inhibit IL-2-driven proliferation of anti-IgM activated B-CLL cells. Furthermore, while IL-4
strongly up-regulated the expression of CD23 on anti-IgM-activated leukaemic B cells, IL-13 only marginally increased it. Finally, IL-13, in contrast to IL-4, did not prevent the entry of B-CLL cells into apoptosis.
Thus IL-13 and IL-4 display comparable effects on anti-CD40-activated B-CLL cells, which are blocked by anti-IL-4 receptor (IL-4R) monoclonal
antibodies. However, ***IL*** - ***13*** -dependent effects
are absent or inefficient in non-activated or anti-IgM-activated B-CLL cells. This suggests that such cells may lack functional IL-13 receptors, though IL-13R and IL-4R on B-CLL cells share a common component.

=> d l24 bbb abs 1-11

L24 ANSWER 1 OF 11 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001167427 MEDLINE
DOCUMENT NUMBER: 21166040 PubMed ID: 11269532
TITLE: Differential in vitro effects of IL-4, IL-10, and IL-13 on pro-inflammatory cytokine production and fibroblast proliferation in rheumatoid synovium.
AUTHOR: Morita Y; Yamamura M; Kawashima M; Aita T; Harada S; Okamoto H; Inoue H; Makino H
CORPORATE SOURCE: Department of Medicine III, Okayama University Medical School, Japan
SOURCE: RHEUMATOLOGY INTERNATIONAL, (2001 Feb) 20 (2) 49-54.
Journal code: 8206885. ISSN: 0172-8172.
PUB. COUNTRY: Germany, Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

Rheumatoid arthritis (RA) is a severe expression of pro-inflammatory cytokine and cytokine-mediated fibroblast growth. IL-4, IL-10, and IL-13
[illegible]

TNF-alpha reduction. The IL-1 receptor antagonist was enhanced by IL-4 and
IL-13, but only slightly enhanced by IL-10. Spontaneous interferon-gamma
secretion was diminished by IL-4 and IL-10 but not by IL-13. Addition of
anti-IL-10 neutralizing antibody to RA synovial tissue cells resulted in a substantial increase in IL-1beta and TNF-alpha levels, whereas neither anti-IL-4 nor anti- ***IL*** - ***13*** ***antibody*** had a significant effect. IL-1beta-stimulated proliferation of RA synovial fibroblast cell lines was inhibited by IL-4 and IL-13, but not by IL-10. IL-4 was over tenfold more effective than IL-13. These results suggest that IL-4, IL-10, and IL-13 all have the therapeutic potential to regulate the disease activity mediated by proinflammatory cytokines in RA, but each
cytokine may have different potencies.

L24 ANSWER 2 OF 11 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2000-431587 [37] WPIDS
DOC. NO. CPI: C2000-131254
TITLE: New polynucleotide encoding an interleukin-13 (IL-13) binding chain of an IL-13 receptor for treating IgE-mediated conditions, such as atopy, asthma, Grave's disease and inflammatory conditions of the lung.
DERWENT CLASS: B04 D16
INVENTOR(S): COLLINS, M; DONALDSON, D; FITZ, L; NEBEN, T; WHITTERS, M
J; WILLS-KARP, M; WOOD, C
PATENT ASSIGNEE(S): (GEMY) GENETICS INST INC; (UYJO) UNIV JOHNS HOPKINS
COUNTRY COUNT: 83
PATENT INFORMATION:

PATENT NO KIND DATE WEEK LA PG

WO 2000036103 A1 20000622 (200037)* EN 60
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL
OA PT SD SE SL SZ TZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE
GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG
MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG
UZ VN YU ZW
AU 2000021775 A 20000703 (200046)
EP 1141286 A1 20011010 (200167) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT
RO SE SI
BR 9916209 A 20011226 (200206)
CN 1352686 A 20020605 (200261)

APPLICATION DETAILS:

PATENT NO KIND APPLICATION DATE

WO 2000036103 A1 WO 1999-US29493 19991213
AU 2000021775 A AU 2000-21775 19991213
EP 1141286 A1 EP 1999-966166 19991213
WO 1999-US29493 19991213
BR 9916209 A BR 1999-16209 19991213
WO 1999-US29493 19991213
CN 1352686 A CN 1999-815591 19991213

FILING DETAILS:

PATENT NO KIND PATENT NO

AU 2000021775 A Based on WO 2000036103
EP 1141286 A1 Based on WO 2000036103
BR 9916209 A Based on WO 2000036103

PRIORITY APPLN. INFO: US 1998-212335 19981214
AN 2000-431587 [37] WPIDS
AB WO 200036103 A UPAB: 20000807
NOVELTY - A polynucleotide comprising a nucleotide sequence that encodes
an interleukin-13 binding chain (IL-13bc) or fragment, of IL-13 receptor,
is new.
DETAILED DESCRIPTION - The polynucleotide comprises a nucleotide
sequence that is:
(a) nucleotides 256 to 1404 of a 1525 murine nucleotide sequence, given in the specification;
(b) nucleotides 103 to 1242 of a 1369 human nucleotide sequence, [illegible]

[illegible]
the [illegible]

(III) amino acids 257 to 383 of (I);
(IV) 380 amino acids, given in the specification;
(V) amino acids 26 to 341 of (IV); or
(VI) amino acids 367 to 380 of (IV); or
(VII) fragments of (I) to (VI) having IL-13 receptor binding chain activity;
(4) a protein produced by (2);
(5) a composition comprising an antibody that reacts with (3);
(6) identifying an inhibitor of IL-13 binding to the IL-13 receptor (IL-13R) comprising:
(i) combining (2) with IL-13 or a fragment to form a first binding mixture;
(ii) measuring binding between the protein and IL-13 or fragment;
(iii) combining a compound with the protein and IL-13 or fragment to
form a second binding mixture;
(iv) measuring the amount of binding; and
(v) comparing the binding in the first binding mixture with the binding in the second binding mixture, where the compound inhibits IL-13
binding to IL-13R when there is a decrease in the binding of the second binding mixture;
(7) an inhibitor identified by (6);
(8) inhibiting binding of IL-13 to IL-13R in a mammal comprising administering (7), (3) or (5);
(9) a polynucleotide comprising a nucleotide sequence that encodes a
peptide or protein with an amino acid sequence of (3);
(10) treating an IL-13-related condition in a mammal by administering
(3) or an IL-13 antagonist;
(11) potentiating IL-13 activity comprising combining a protein with IL-13 activity with (3) and contacting the combination with a cell expressing a chain of IL-13R other then IL-13bc; and
(12) inhibiting interaction of IL-13 with IL-13bc in a mammal by administering IL-13 antagonist.
ACTIVITY - Antiallergic; antiinflammatory; antiasthmatic; dermatological; immunosuppressive; antithyroid; cytostatic.
Male A/J mice were immunized intraperitoneally and challenged intratracheally with soluble ovalbumin. The allergic phenotype was assessed 4 days after the antigen challenge. Blockade of IL-13 was performed 24 hours before the allergen challenge by systemic administration of soluble IL-13bc-IgGFc fusion protein which binds to and
neutralizes IL-13. Challenge of allergen-immunized mice resulted in significant increases in airway responsiveness to acetylcholine. Blockade
of IL-13 resulted in complete reversal of the established allergen-induced
airway hyper responsiveness, showing that asthma may be treated.
MECHANISM OF ACTION - IL-13 inhibitor.
USE - For identifying and producing an IL-13bc protein that can inhibit the binding of IL-13 to an IL-13 receptor and treat an IL-13-related condition such as an IgE-mediated condition. Atopy, allergic conditions, asthma, immune complex diseases, lupus, nephritis, thyroiditis, Grave's disease or inflammatory conditions of the lung can be
treated. For potentiating IL-13 activity (all claimed). Cancer may be treated. Macrophage activation is enhanced allowing use in vaccination and treatment of mycobacterial, intracellular organisms, or parasitic infections.
Dwg.0/4

L24 ANSWER 3 OF 11 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000454825 MEDLINE
DOCUMENT NUMBER: 20363565 PubMed ID: 10903803
TITLE: Interleukin-13 and IgE production in rat experimental schistosomiasis.
AUTHOR: Cetre C; Pierrot C; Maire E; Capron M; Capron A; Khalife J
CORPORATE SOURCE: Institut Pasteur de Lille, INSERM U 167, 1, rue du Pr
Calmette, BP 245, 59019 Lille Cedex, France.
SOURCE: EUROPEAN CYTOKINE NETWORK, (2000 Jun) 11 (2) 241-49.
Journal code: 9100879. ISSN: 1148-5493.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200009
ENTRY DATE: Entered STN: 20001005
Last Updated on STN: 20001005
Entered Medline: 20000925
AB We have previously demonstrated in rat experimental schistosomiasis an
upregulation of IL-4 expression at the mRNA and protein levels which could
[illegible] increased IgE production observed [illegible]

[illegible] infected rats as compared to those from non-infected [illegible] animals. These findings show that, similarly to IL-4, IL-13 is implicated
[illegible]

antibodies showed significant decrease in the IgE levels.
Moreover, administration of IL-13 enhanced total IgE levels. These results
demonstrate the implication of IL-4 and IL-13 in vivo in IgE production,
and provide a relevant animal model for a better understanding of the role
of IL-4 and IL-13 in humans.

L24 ANSWER 4 OF 11 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001097379 EMBASE
TITLE: Differential in vitro effects of IL-4, IL-10, and IL-13 on proinflammatory cytokine production and fibroblast proliferation in rheumatoid synovium.
AUTHOR: Morita Y.; Yamamura M.; Kawashima M.; Aita T.; Harada S.;
Okamoto H.; Inoue H.; Makino H.
CORPORATE SOURCE: M. Yamamura, Department of Medicine III, Okayama University
Medical School, 2-5-1 Shikata-cho, Okayama 700-8558, Japan.
yamamura@med.okayama-u.ac.jp
SOURCE: Rheumatology International, (2000) 20/2 (49-54).
Refs: 38
ISSN: 0172-8172 CODEN: RHINDE
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
031 Arthritis and Rheumatism
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The purpose of this study was to compare the potential of interleukin-4
(IL-4), IL-10, and IL-13 to interrupt two major inflammatory pathways in
rheumatoid arthritis (RA), i.e., overexpression of proinflammatory cytokines and cytokine-mediated fibroblast growth. IL-4, IL-10, and IL-13
were all able to significantly inhibit the production of IL-1.beta., tumor necrosis factor-.alpha. (TNF-.alpha.), IL-6, and IL-8 by freshly isolated RA synovial tissue cells; IL-10 was most effective in terms of IL-1.beta. and TNF-.alpha. reduction. The IL-1 receptor antagonist was enhanced by
IL-4 and IL-13, but only slightly enhanced by IL-10. Spontaneous interferon-.gamma. secretion was diminished by IL-4 and IL-10 but not by
IL-13. Addition of anti-IL-10 neutralizing antibody to RA synovial tissue
cells resulted in a substantial increase in IL-1.beta. and TNF-.alpha. levels, whereas neither anti-IL-4 nor anti- ***IL*** - ***13*** ***antibody*** had a significant effect. IL-1.beta.-stimulated proliferation of RA synovial fibroblast cell lines was inhibited by IL-4 and IL-13, but not by IL-10; IL-4 was over tenfold more effective than IL-13. These results suggest that IL-4, IL-10, and IL-13 all have the therapeutic potential to regulate the disease activity mediated by proinflammatory cytokines in RA, but each cytokine may have different potencies.

L24 ANSWER 5 OF 11 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97350814 MEDLINE
DOCUMENT NUMBER: 97350814 PubMed ID: 9207190
TITLE: Expression of recombinant rat interleukin-13 (IL-13) and generation of a neutralizing rat IL-13 antiserum.
AUTHOR: Lakkis F G; Cruet E N; Nassar G M; Badr K F; Pascual D W
CORPORATE SOURCE: Renal Division, Emory University School of Medicine and
Veterans Affairs Medical Center, Atlanta, Georgia 30033, USA. flakkis@emory.edu
CONTRACT NUMBER: AI 40288 (NIAID)
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1997
Jun 27) 235 (3) 529-32.
Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 19970805
Last Updated on STN: 19970805
Entered Medline: 19970724
AB Using baculoviral and bacterial systems, we expressed biologically active
recombinant rat IL-13 and generated neutralizing rat IL-13 antiserum.
Recombinant rat IL-13 produced by baculovirus-infected insect cells stimulated proliferation of TF-1 premyeloid cell line and induced

[illegible] supernatants of insect cells infected with recombinant baculovirus
carrying the rat IL-13 cDNA. The availability of recombinant rat IL-13 [illegible]

L24 ANSWER 6 OF 11 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 97222301 MEDLINE
DOCUMENT NUMBER: 97222301 PubMed ID: 9069451
TITLE: Interleukin-13 inhibits cytokine secretion by blood monocytes from patients with IgA nephropathy.
AUTHOR: Matsumoto K
CORPORATE SOURCE: Second Department of Internal Medicine, Nihon University School of Medicine, Itabashi-ku, Tokyo, Japan.
SOURCE: NEPHRON, (1997) 75 (3) 295-302.
Journal code: 0331777. ISSN: 0028-2766.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 19970724
Last Updated on STN: 19970724
Entered Medline: 19970715
AB In this study we have examined the effects of recombinant human interleukin (IL)-13 on peripheral blood monocytes (PBM) from patients with IgA nephropathy (IgAN). Significantly increased spontaneous and lipopolysaccharide (LPS)-stimulated secretion of tumor necrosis factor-alpha (TNF) and IL-8 was determined in PBM cultures of IgAN patients compared to those of normal controls. In the present study, IL-13 inhibited the spontaneous as well as the LPS-stimulated cytokine secretion of PBM in IgAN. Significant inhibitory effect of IL-13 was observed in cultures of PBM from IgAN patients as well as from normal persons. When both LPS and anti-***IL*** + ***13*** + ***antibody*** were added together to the PBM, a further increase of LPS-enhanced secretion of cytokines occurred. Taken together, these results indicate that IL-13 down-regulates the secretion of TNF and IL-8 from IgAN PBM.

L24 ANSWER 7 OF 11 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 1998004375 MEDLINE
DOCUMENT NUMBER: 98004375 PubMed ID: 9346389
TITLE: Interleukin 10 and interleukin 13 synergize to inhibit vascular permeability factor release by peripheral blood mononuclear cells from patients with lipoid nephrosis.
AUTHOR: Matsumoto K; Ohi H; Kanmatsuse K
CORPORATE SOURCE: 2nd Department of Internal Medicine, Nihon University School of Medicine, Tokyo, Japan.
SOURCE: NEPHRON, (1997) 77 (2) 212-5.
Journal code: 0331777. ISSN: 0028-2766.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19980109
Last Updated on STN: 19980109
Entered Medline: 19971126
AB It has been proposed that a vascular permeability factor (VPF) is involved in the pathogenesis of lipoid nephrosis (LN). There is now increasing evidence that interleukin 10 (IL-10) and interleukin 13 (IL-13) have regulatory effects on cytokine production by activated macrophages. These results prompted us to study the effects of recombinant human IL-10 and IL-13 on VPF secretion in LN. In the present study, we demonstrate that the regulatory cytokines IL-10 and IL-13 are potent inhibitors of the VPF activity of activated peripheral blood mononuclear cells. Each cytokine was found to suppress VPF secretion in a dose-dependent fashion. Moreover, importantly, the combination of the cytokines was found to give a potent synergistic suppression of VPF by concanavalin A-activated peripheral blood mononuclear cells from patients with LN. When both anti-IL-10 and anti-***IL*** + ***13*** + ***antibodies*** were added together to the peripheral blood mononuclear cells, a further increase of concanavalin A-enhanced secretion of VPF occurred. These data establish IL-10 and IL-13 as potent inhibitors of VPF activity and suggest their utility in controlling cytokine-VPF-mediated responses such as occur in LN patients with nephrotic syndrome.

L24 ANSWER 8 OF 11 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 97339858 MEDLINE
DOCUMENT NUMBER: 97339858 PubMed ID: 9212111
TITLE: Interleukin-13 (IL-13) pathway in human fetal gut development: IL-13 induces gut hypertrophy and interacts with IL-4 in modulating epithelial cell proliferation.
AUTHOR: Minty A; Chalon P; Guillemot J C; Kaghad M; Liauzun P; Magazin M; Miloux B; Minty C; Ramb S; Vita N; Caput D; Ferrara P
CORPORATE SOURCE: Sanofi Recherche, Centre de Recherche de Toulouse, France.
SOURCE: AMERICAN JOURNAL OF RESPIRATORY CELL AND MOLECULAR BIOLOGY, (1997 Jul) 17 (1) 20-8.
Journal code: 8917225. ISSN: 1044-1549.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 19970828
Last Updated on STN: 19970828
Entered Medline: 19970814
AB Interleukin-13 (IL-13) is a recently cloned cytokine that has been shown to have a number of activities in common with IL-4. Both cytokines bind to a receptor complex containing the IL-4 receptor alpha-chain and, in addition, IL-13 binds to a unique receptor alpha-chain. We have investigated the role of IL-13 in human fetal gut development and compared its activities with those of IL-4. Using in situ hybridization, we have shown that IL-13 mRNA is expressed in the developing human fetal gut, with a pattern of expression similar to that of IL-4 mRNA. Both IL-13 and IL-4 induced a dose-dependent hypertrophy of human fetal gut explants in organ culture. Histologic examination revealed that this hypertrophy was due to epithelial cell proliferation. Both cytokines induced a similar degree of hypertrophy, and the effects of IL-13 and IL-4 were not additive. Immunohistochemical staining revealed that IL-13 receptor alpha-chain is expressed in the developing human fetal gut, with a pattern of expression similar to that of IL-4 receptor alpha-chain. Both receptor alpha-chains were expressed on epithelial cells. These observations suggest that IL-13 and IL-4 may play a role in human fetal gut development by inducing epithelial cell proliferation.

L24 ANSWER 9 OF 11 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 96256149 MEDLINE
DOCUMENT NUMBER: 96256149 PubMed ID: 8675220
TITLE: In vivo treatment with anti-interleukin-13 antibodies significantly reduces the humoral immune response against an oral immunogen in mice.
AUTHOR: Bost K L; Holton R R; Cain T K; Clements J D
CORPORATE SOURCE: Department of Microbiology and Immunology, Tulane University Medical Center, New Orleans, LA 70112, USA.
CONTRACT NUMBER: AI28835 (NIAID)
AI32976 (NIAID)
SOURCE: IMMUNOLOGY, (1996 Apr) 87 (4) 533-41.
Journal code: 0374672. ISSN: 0019-2805.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199608
ENTRY DATE: Entered STN: 19960822
Last Updated on STN: 19960822
Entered Medline: 19960809
AB Interleukin-13 (IL-13) is a cytokine which significantly enhances the proliferation and differentiation of B lymphocytes. We therefore evaluated its role in the formation of a humoral immune response in vivo. Upon oral immunization with the B subunit of Escherichia coli heat-labile enterotoxin (LT-B), rapid up-regulation of IL-13 mRNA expression in the mesenteric lymph nodes of LT-B intubated mice occurred. This result suggested that IL-13 might be involved in the formation of a mucosal antibody response against LT-B if this cytokine was in fact secreted. To test this possibility, the coding region for murine IL-13 was cloned into the pET-AG-1 expression vector. Recombinant murine IL-13 was purified from bacterial lysates and used as an immunogen to produce polyclonal anti-***IL*** + ***13*** + ***antibodies*** in BALB/c mice. Administration of anti-***IL*** + ***13*** + ***antibodies*** demonstrated decreased expression of IL-4 and IL-13 mRNA and decreased IL-4 secretion when compared to controls. Together these results demonstrate an important role for IL-13 in the formation of a humoral immune response at mucosal surfaces.

L24 ANSWER 10 OF 11 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 95339858 MEDLINE
DOCUMENT NUMBER: 95339858 PubMed ID: 7614976
TITLE: Involvement of interleukin (IL)-13, but not IL-4, in spontaneous IgE and IgG4 production in nephrotic syndrome.
AUTHOR: Kimata H; Fujimoto M; Furusho K
CORPORATE SOURCE: Department of Pediatrics, Kyoto University Hospital, Japan.
SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (1995 Jun) 25 (6) 1497-501.
Journal code: 1273201. ISSN: 0014-2980.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199508
ENTRY DATE: Entered STN: 19950905
Last Updated on STN: 19950905
Entered Medline: 19950822
AB Nephrotic syndrome (NS) is a renal disease characterized by proteinuria and hypoalbuminemia. In NS patients without any allergic disease, serum IgE and IgG4 levels were selectively increased, and peripheral blood mononuclear cells (MNC) spontaneously produced IgE and IgG4. T cells produced interleukin (IL)-13 spontaneously, and B cells constitutively expressed IL-13 receptors (IL-13R). In addition, T cells stimulated surface IgE-negative (sIgE-) and sIgG4- B cells to produce IgE and IgG4, respectively, and IgE and IgG4 production was specifically blocked by anti-***IL*** + ***13*** + ***antibody*** (Ab). MNC from atopic dermatitis (AD) patients also produced IgE and IgG4 spontaneously. However, in AD patients, T cells spontaneously produced IL-4, but not IL-13, and B cells constitutively expressed IL-4R, but not IL-13R. T cells stimulated sIgE- and sIgG4- B cells to produce IgE and IgG4, respectively, and the production was specifically blocked by anti-IL-4 Ab. On the other hand, sIgE+ and sIgG4+ B cells from both NS and AD patients spontaneously produced IgE and IgG4, respectively, and this production was not affected by T cells, anti-IL-4 Ab, or anti-IL-13 Ab. These results indicate that IL-13 is involved in the enhanced production of IgE and IgG4 in NS, while IL-4 is involved in these responses in AD.

L24 ANSWER 11 OF 11 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 95244773 MEDLINE
DOCUMENT NUMBER: 95244773 PubMed ID: 7727691
TITLE: Interleukin-13 gene expression by malignant and EBV-transformed human B lymphocytes.
AUTHOR: Fior R; Vita N; Raphael M; Minty A; Maillot M C; Crevon M C; Caput D; Biberfeld P; Ferrara P; Galanaud P; +
CORPORATE SOURCE: INSERM U131, Clamart, France.
SOURCE: EUROPEAN CYTOKINE NETWORK, (1994 Nov-Dec) 5 (6) 593-600.
Journal code: 9100879. ISSN: 1148-5493.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199506
ENTRY DATE: Entered STN: 19950608
Last Updated on STN: 19980206
Entered Medline: 19950601
AB Expression of the IL-13 gene in malignant tissues from 26 human B-cell lymphoid malignancies was analyzed by reverse transcriptase-polymerase chain reaction (RT-PCR). A positive signal was detected in 16 cases, which included high grade B lymphomas, follicular lymphomas and B cell chronic lymphocytic leukemias. IL-13 mRNA was also detected in the 9 malignant B cell lines and in the 6 lymphoblastoid cell lines tested, as well as in [illegible] ***IL*** + ***13*** + ***antibodies*** to affect the growth of malignant B cells. Taken together, these results show that both malignant and EBV-transformed B lymphocytes, either [illegible]

as
well as on the in vivo behaviour of B lymphoid malignancies

- - log off
ALL I# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y) N HOLD:x
SDN INTERNATIONAL LOGOFF AT 20:35:43 ON 19 OCT 2002

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=ADJ			
L11	(l10 or l9) and l4	0	L11
L10	zhang-jian.in.	8	L10
L9	metcalf-donald.in.	16	L9
L8	hilton-douglas.in.	0	L8
L7	nicola-nicos.in.	0	L7
L6	wilson-tracy.in.	0	L6
L5	L1 adjn10 antibod$3	0	L5
L4	L1 same antibod$3	94	L4
L3	L1 with antibod$3	40	L3
L2	L1 and antibod$3	440	L2
L1	il-13 or il 13 or interleukin1-13 or interleukin 13	480	L1

END OF SEARCH HISTORY